THE PROBLEM OF TAKE-ALL DISEASE IN CONTINUOUS CEREAL-GROWING IN SOUTH-EAST SCOTLAND

by

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ABSTRACT

The literature of the ecological and agricultural aspects of take-all disease caused by Gaeumannomyces graminis var tritici is reviewed.

Assessment of the disease in a long-term spring barley trial from its 3rd to 12th years revealed that, although it developed in the accepted take-all decline pattern, the onset of decline was delayed. Incidence was low until the 9th season, rose to a maximum of 80% in the 11th and decreased to about 45% in the 12th year. Applications of 50, 100 and 150 kg/ha of nitrogen fertiliser decreased incidence but different methods of ploughing had more variable results: until the 6th year in barley take-all was less prevalent in the deep- and unploughed compared with the shallow- and chisel-ploughed treatments, but subsequently only direct-drilling suppressed disease. At the peak of infection there was no difference in incidence between cultivation treatments. Severity of infection increased slightly from the 3rd to 12th crops but was always low.

A technique was devised to predict the development of disease in different soil types. When soil from the long-term barley trial was assayed the occurrence of infection was similar in some ways to the field pattern. An experiment using the technique compared infection in four soils: a sand, a sandy loam, a clay loam and a clay, each at four cropping histories. Although infection patterns peaked and declined in all soils their disease development differed with time and cropping history. Inoculation increased level of infection but did not change the pattern. Assaying field soil samples collected in 1975 and 1976 revealed a wide range of disease patterns.

Evolvement of a technique to assess the virulence of field

populations of the take-all fungus showed that virulence was not correlated with soil texture or cereal history but might be affected by previous cultivation technique.

The theory and practical problems of the assay techniques and their relevance to contempory agriculture are discussed.

Incidence of the take-all fungus was found to be widespread in the soils of South-East Scotland yet loss of grain yield from infection appeared to be small unless other adverse predisposing factors were present.

This thesis was composed by myself and describes work which I carried out between October, 1974 and September, 1977.

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Introduction

Take-all is a fungal disease of the roots of barley, wheat and oats. These crops are considered to be most at risk when grown in monoculture. Although research on take-all began in the early years of this century (Nilsson, 1969), the basis of modern knowledge of this disease is work by S D Garrett in Australia in the 1930s when wheat grown on light sandy soil after the clearance of scrub vegetation suffered great losses. Since then the general expansion in cereal acreage has resulted in the increase of both the disease and related research. The growth characteristics of the causal fungus and symptoms of take-all are well documented (Brown and Hornby, 1969, 1971; Holland and Fulcher, 1971; Weste, 1972; Clarkson, Drew, Ferguson and Sanderson, 1975; and Holden, 1976). Previous work was surveyed by Nilsson in 1969. As the fungus can survive only on infected debris or a few weed grass species in absence of a susceptible crop, rotation of cereals with other crops kept the disease at an unremarkable level in Britain until the intensification of cereal-growing during the last war. Recently the phenomenon of take-all decline has been noted in continuously grown cereals in England (Cox and Slope, 1963, and Shipton, 1967) and in the Dutch polders (Gerlagh, 1968), although evidence for a decline phase was unwittingly presented by Glynne (1935) and Zogg (1951). In 1973, Shipton, Cook and Sitton recorded an area of soil in the United States with apparently innate antagonism to the serious development of the disease.

In South-East Scotland no instance of take-all decline has yet been documented. This is at least partly explained by the relatively late intensification of cereals and the generally cautious nature of the farmers of this area, for runs of more than five or six cereal crops

are uncommon; however there has been a rapid increase in cereal-growing in the last two decades, particularly in barley, and thus a potential risk of substantial yield losses from take-all. There are no published data on the levels of take-all found in this area apart from those in a general survey of Scottish cereal disease incidence by Richardson (1972) who claimed an overall figure of about 1% for'take-all and other whiteheads'. In response to the dearth of information about cereal monoculture in the East of Scotland area a long-term experiment was set up in October, 1965, at South Road Field, Langhill Farm, Midlothian to investigate the effect of different cultivations and nitrogen levels on barley. During the third and subsequent seasons the levels of take-all were assessed and found to differ markedly from the accepted pattern, that is, a peak of infection from the 4th to 6th years (Shipton, 1967) followed by a decline.

After reviewing previous studies relating to the occurrence of take-all, factors affecting its incidence and approaches to its control, this work records and discusses the development of take-all in the South Road Experiment until the twelfth crop. It also examines different soils in the South-East of Scotland in relation to their potential for take-all development and assesses the possibility of predicting take-all patterns in continuously grown cereals based on a knowledge of soil, field history and fungal factors, particularly isolate virulence.

Introductory Literature

Take-all disease is caused by the fungus Gaeumannomyces graminis (Sacc) Arx and Olivier, previously known as Ophiobolus graminis Sacc (Arx and Olivier, 1952). In a detailed examination of the taxonomy of G. graminis, Walker (1972) recognised three varieties: var tritici Walker, the wheat take-all fungus which also attacks barley

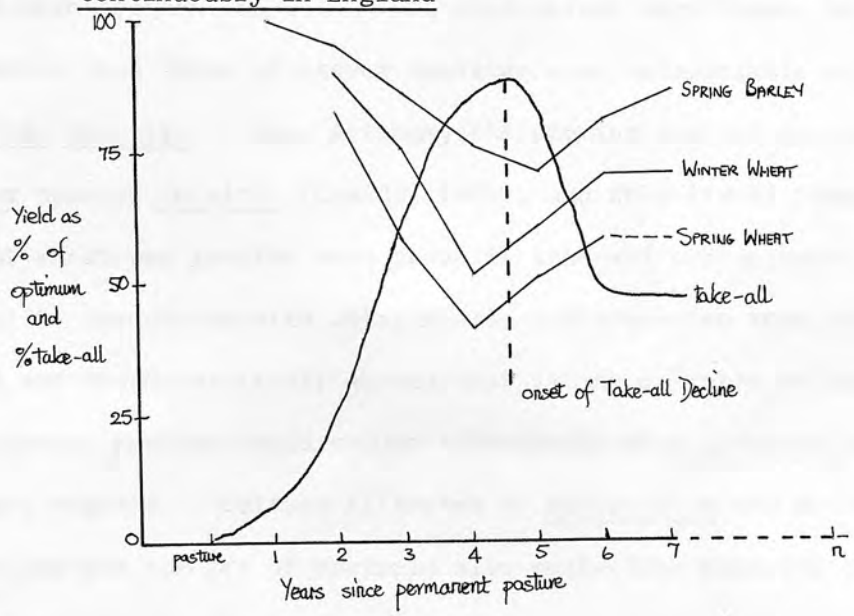
and other members of the Gramineae but not oats; var avenae Turner, which attacks oats as well as other cereals and is responsible for a patch disease of turf; and var graminis, the 'type' fungus, the least pathogenic of the three varieties, responsible for brown sheath rot of rice.

The symptoms, effects and basic ecology of the disease have been extensively researched and were reported in detail by Nilsson in 1969. Since then the aspect of ecology which has been investigated most is biological control and especially the phenomenon known as take-all decline. Although Glynne reported a reduction of the disease in wheat after several years in 1935 at Rothamsted Experimental Station, take-all decline was first described as such by Slope and Cox in 1963. They found that in a fourth consecutive wheat crop, take-all declined and yield increased to a level similar to that normally found in the continuous wheat crop in Broadbalk Field at Rothamsted. They concluded that there was a factor suppressing take-all which they called take-all decline (TAD). This pattern continued in the fifth and seventh consecutive wheat crops (Cox, 1966). In 1973 Brown, Hornby and Pearson described the phenomenon as it is now generally understood, i.e. that take-all attack is at a maximum after two or three consecutive cereal crops, decreases to the fifth or sixth crop and then remains fairly constant. Doling and Batts (1960) observed twice as much take-all infection in a winter wheat crop which had followed a spring wheat - peas - spring wheat rotation than in one following three years winter wheat, but they did not comment on this. Gerlagh (1968) recorded marked TAD in continuous wheat grown on reclaimed polder, and Shipton (1967) confirmed, from a survey of continuous cereal crops outside Rothamsted, the

existence of TAD in winter wheat, spring wheat and barley but stressed that levels of disease decline and yield increase varied with weather. Lester and Shipton (1967), using a wheat-seedling test, noted considerable difference in the ability of various soils with contrasting cropping histories to reduce disease incidence after known quantities of inoculum had been added. Lemaire and Coppenet (1968) found that the severity of take-all decreased in the third or fourth continuous cereal crop and that interruption with a break crop usually caused an increase in attack. They claimed that soils in contact with diseased cereals develop a 'biocoenosis' which is antagonistic to G. graminis. Shipton (1972b) confirmed that TAD in field trials was primarily expressed by reduced severity, thus suggesting antagonism was limiting pathogen growth after infection.

Shipton (1967) expressed the generalised pattern of yields of continuously grown cereals in the form of a graph which is reproduced below.

Generalised pattern of yields of wheat and barley grown continuously in England



Zogg (1951) drew a hypothetical yield curve for continuous cereals grown in the alpine region. He claimed that within two or three years yield falls to 10% of that under good rotation, and that substantial yield recovery to a level of 80% of the original may take 30 to 70 years. The subsequent yield improvement he considered to "merely illustrate the passing away of a (disease) epidemic", (personal communication to Shipton, 1967). Later Zogg (1972) amended the low yield figure after two years of cereals to "50, 30 or 10%, depending on the weather conditions". The discrepancy between Zogg's claims and the findings of Gerlagh (1968) may be ascribed to the fairly sterile nature of newly-drained polders. Even though this enables very marked antagonism to develop, he found antagonism was more easily established in 'older' soils.

With the recognition of TAD, Cox (1963) investigated the antagonistic factor by estimating the populations of Actinomycetes in soils from a cereal-bean experiment and Broadbalk: each field had a distinctive Actinomycete flora and only a small proportion of the species were frequent on both. No conclusions were drawn, but she did observe that three of eleven isolates were antagonistic to G. graminis in vitro. Some Actinomycete strains are antagonistic to Fusarium species in vitro (Lepidi, 1969), and Ehle (1966) found grain yield of wheat was greater when grown in take-all contaminated vermiculite inoculated with antagonistic Actinomycetes than without. Sanford and Broadfoot (1931) showed that living cultures of some fungal and bacterial species could reduce infectivity of G. graminis by different degrees. Culture filtrates of Penicillium and Actinomycetes species and six species of bacteria also controlled severity of G. graminis, but to a lesser extent.

A number of fungi have been found to be antagonistic to

to G. graminis: Trichoderma viride (Lal, 1939; Ludwig and Henry, 1943; Slagg and Fellows, 1947; Skipsna, 1961; Ponomareva, 1968), Penicillium species (Lal, 1939; Slagg and Fellows, 1947; Skipsna, 1961; Mangan, 1967; Ponomareva, 1968), Cladosporium species (Skipsna, 1961; Ponomareva, 1968), some Aspergillus species (Lal, 1939; Slagg and Fellows, 1947); Gliomastix species (Mangan, 1967; Domsch and Gams, 1968), Fusarium culmorum (Lal, 1939), Paecilomyces carneus (Mangan, 1967), Didymella exitialis (Fluck, 1955; Siegle, 1961) and others (Domsch and Gams, 1968). Gerlagh (1968) found three fungal genera present on the Polders were antagonistic to G. graminis in a pot experiment: Gliocladium, Mucor and Chaetomium. Members of the family Mucoraceae (Skipsna, 1961), the order Labyrinthulales (Schmoller and Weigershausen, 1968) and the genus Streptomyces (Domsch and Gams, 1968) were also found to be antagonistic.

Lal (1939) observed that Rhizoctonia solani was antagonistic to G. graminis, but Van der Watt (1965) claimed that R. solani eliminates some of the antagonists to G. graminis and may thus increase its ability to survive. Slagg and Fellows (1947) noted a variation of inhibitory ability of fungi dependent on the substrate on which they were cultured, and Broadfoot (1933b) showed that presence or absence of antagonism on agar plates does not necessarily indicate a similar reaction in the soil.

There has been some interest in Trichoderma viride as a possible antagonist to G. graminis: inoculation of acidified soil with T. viride was found to control R. solani which is responsible for damping-off of citrus seedlings; no control was achieved in acidified soils without T. viride (Weindling and Fawcett, 1936). Although Slagg and Fellows reported that T. viride could penetrate and kill hyphae of G. graminis, Lal found that only some strains of T. viride were inhibitory. No strains isolated by Mangan (1967) were inhibitory.

Fluck (1955) found no evidence of mutual antagonism between Didymella exitialis and G. graminis in vitro but a culture filtrate of D. exitialis strongly inhibited growth of G. graminis on wheat. Siegle (1961) observed that D. exitialis could penetrate and destroy G. graminis hyphae in the rhizosphere.

Gerlagh (1968) attempted to establish the microbial origins of TAD on the East Flevoland Polder. He first distinguished between general antagonism in the form of competition from other micro-organisms without which growth of G. graminis on the host is optimum (Ludwig and Henry, 1943), and specific antagonism which results in TAD. Garrett (1934) was describing the first type of antagonism when he observed that take-all infection was always worst on wheat seedlings grown in soils low in organic matter and bacterial numbers.

Gerlagh suggested five different hypotheses which could explain TAD:

1. G. graminis induces a biological antagonism against itself;
2. G. graminis poisons its own habitat by producing harmful chemicals (staling effect);
3. G. graminis loses its virulence after a heavy attack;
4. plentiful supply of nutrients after a bad harvest caused by take-all will enable the next crop to resist G. graminis better;
5. after a heavy attack plants die earlier than normal, leaving less infected material as plants remain smaller, and this is exposed to competitive saprophytism longer than after a light attack. Successive crops are thus exposed to decreasing inoculum levels.

Hypotheses 3 and 5 were discarded when it was found that antagonism was induced even when fresh, virulent inoculum was used for each cycle of plants in the soil. Hypothesis 4 was disproved

by the fact that antagonism arose when the amount of fertiliser added to the soil was corrected for the weight of the previously harvested wheat crop. Gerlagh thought that any 'staling' products would be degraded very quickly in a microbiologically active medium, such as soil, and concluded that the first hypothesis was the most probable. Microbial antagonism could be effected by specific competition for nutrients, by hyperparasitism, or by the production of antibiotics, although the latter would have a chemical basis and be similar to staling. Extensive glasshouse experiments led him to conclude that antagonism is probably caused by microbial antibiotics produced by a balanced association of micro-organisms, although he could find no positive evidence against hyperparasitism or specific competition for nutrients, or indeed against the involvement of only one organism. However, he remarked that decline might depend on one antibiotic produced by several organisms. He tentatively suggested that a fungus was implicated. Attempts to isolate the antagonistic factor failed; no differences could be found in the antagonistic and non-antagonistic soils, but perhaps only because of inadequate techniques. Gerlagh explained this failure by observing that as antibiotic properties depend on the environment, the actual capacity of an organism cannot be measured in vitro. The experiments showed that antagonistic soil reduces the growth of G. graminis in its parasitic stage as well as its survival in the saprophytic stage. If this is so, a gradual decrease in incidence of the disease in the field, as measured by the percentage of roots infected, as well as a decrease in severity, ought to be observed; as the growth and survival of the fungus in any season depends on environmental conditions, this point could be very difficult to demonstrate in vivo over a short period.

In 1970 Lapierre, Lemaire, Jouan and Molin reported that virus particles of cubic symmetry were isolated from weakly pathogenic strains of G. graminis. The virus particles were lost during the formation of ascospores, which germinated to produce highly pathogenic strains. Rawlinson, Hornby, Pearson and Carpenter (1973) found isometric virus-like particles (VLP) of 35 and 27 nm diameter in mycelium of G. graminis var tritici and var avenae. More than half of 145 isolates from cereals after two to twelve consecutive susceptible crops contained either 35 or 27 nm particles. VLP were not confined to G. graminis from soils exhibiting TAD nor consistently associated with weak pathogenicity or with any other growth or morphological character. Isolates with one kind of particle were mostly more pathogenic, and those with both kinds less pathogenic, than isolates without VLP. The proportion of isolates with 27 and 35 nm particles increased progressively in samples from different consecutive crops during the first nine years of cropping, then decreased. VLP could be transmitted through anastomosis with infected isolates. They concluded by stating that they did not know whether VLP can affect decline by influencing survival in the soil, and that there may be viruses responsible for decline as yet undetected by electron microscopy.

In more recent work Wong (1975) reported that two isolates of G. graminis var graminis, G1 and G5, could protect wheat roots against two var tritici isolates, T2 and T1, respectively. As Rawlinson and Muthyalu (1975) found 35 nm VLP in G1 and T1, they suggested a virus-related factor could be implicated in this form of biological control.

Siegle (1961) found that a culture filtrate of D. exitialis, containing five amino acids and "two other ninhydrin-positive

substances", reduced the pathogenicity of G. graminis to about 40% but had no effect on runner hyphae. This could be interpreted on the basis that D. exitialis was supplying the fungus with nutrients, thus reducing the necessity for it to parasitise the vascular tissue of the host. If this was so, TAD may be caused by the development of a particular microbial population affecting nitrogen nutrition. Brown, Hornby and Pearson (1973) investigated the relationship of nitrogen with TAD and although they found a relationship between total nitrogen in bulk soil and maximum disease they regarded the association as 'fortuitous'. They suggested, however, that TAD operates through changes in the soil microflora in response to the progress of the disease and that these modify the root nutritionally, limiting the disease. One possible mechanism is through changes in the nitrate-nitrogen: ammonium-nitrogen ratio in the rhizosphere, an idea based on work by Hornby and Goring (1972). Their only evidence to support this was the finding that the ratio was minimal in the rhizosphere of wheat seedlings growing in soil supporting maximum disease.

Subsequently Brown (1974) stated there was no correlation between disease incidence and ratio of NH_4^+ and NO_3^- ions in the wheat rhizosphere. She did, however, describe the effect of sterile soil extracts from different cereal sequences on the development of take-all: using added inoculum it was found that seedlings were most infected when grown with extract from the third year of a sequence and less with extracts from later years, which corresponds to a decline pattern. A similar pattern of incidence occurred, but with much less infection, when non-sterile extracts obtained by filtration through a 3 μm millipore were used. By repetition of these experiments with addition of potassium nitrate or ammonium sulphate it was found that ammonium sulphate with or without potassium nitrate depressed infection of seedlings grown on sterile extracts from the third and sixth years

and with non-sterile extract from the third year only.

The implications of this report are that, firstly, the factor responsible for the decline pattern in this instance was neither living nor heat-sensitive, and secondly, a microbial organism or denaturable substance caused a further overall decrease in infection. Furthermore, the response to nitrogen and nitrogen-ion type was equivocal.

That there is often found a non-specific antagonism in some agricultural soils has been acknowledged for many years (eg Garrett, 1934). The role of micro-organisms at the root surface encompasses not only antagonism to other micro-organisms, whether directly or indirectly, but also competition with the host root itself, as Morrison (1976) found when soil sterilisation increased cereal yields and decreased the amount of nitrogen required.

In 1973 Pope and Jackson investigated the TAD factor by determining the infectivity of organic debris extracted from wheat-field soils. Their findings coincided with the by-then expected pattern of a peak in the second or third crop and a decline by the fifth successive crop. They also found the factor was transmissable in whole soil, or its suspension, to non-decline soil. In decline soil, hyphal responses to wheat root exudates were significantly reduced from a radius of up to 8 mm to a maximum of 5 mm from the root surface, but they could not decide whether this was caused by direct action of microbial metabolites on the fungus or microbial intervention in its response to the root exudate.

Pope and Hornby (1975) found that as little as 0.001% by weight of a decline soil could transmit a factor which depressed infection by 50% in seedling assays, but the effect was temporary in a field experiment: infection increased to the level in non-decline

soil within ten weeks. The factor was contained in the 0.2 to 2.0 µm fractions but not the coarser fractions of leachates of decline soil and had the following characteristics: it moved downward in water; was most effective when placed above the seed layer and least effective when placed below; lost some effectiveness when added after 14 days of seedling growth in non-decline soil; and was variable in its effect when added up to 4 weeks before planting. It could also be eliminated by heating moist soil for thirty minutes at 70°C, but not at 60°, which implies that the factor was biological. They did not claim that the factor in their experiments was the same as the cause of TAD.

Shipton, Cook and Sitton (1973) reported a suppressive factor in soils under long term wheat culture in Eastern Washington not found in land converted from native vegetation to intensive wheat cropping, when severe outbreaks of take-all occurred. The antagonistic properties of the long-term wheat soil were destroyed by methyl bromide fumigation and by steam pasteurisation at 60°C but could be reinstated by mixing with 1% antagonistic soil in glass-house experiments, although not completely in field plots. The development of antagonism apparently occurred without the interim severe attack. The response to pasteurisation at 50° to 60° led them to conclude that the factor was not linked with Actinomycetes and spore-forming bacteria, and suggested the involvement of soil fungi or non-sporing bacteria.

The ecology of Gaeumannomyces graminis

Garrett (1936) described the life cycle of G. graminis as alternating between a parasitic 'ascending' phase, and a saprophytic 'declining' phase. With this changing ecological role in the soil its environmental requirements change accordingly.

Garrett realised that in the saprophytic stage the fungus is exposed to competition with other soil micro-organisms, but it would not be wise to assume that G. graminis fills its parasitic ecological niche without competition of some description, for example, the avirulent graminaceous parasite, Phialophora radicicola (Deacon, 1973a).

Garrett (1950) classified G. graminis as a root-inhabiting fungus which, while capable of growth in axenic culture, is in nature invariably found in association with susceptible host tissues and is thus regarded as an ecologically obligate parasite. After invasion of a host by G. graminis weaker parasites, such as Aureobasidium bolleyi and some species of Fusarium and Pythium, may colonise the root; in turn these are followed by saprophytes which actively colonise the remaining dead tissue.

The incidence of take-all in the field, as measured by the percentage of plants infected, depends not only on the distribution of foci of inoculum in the soil but on the rate of spread from plant to plant. Severity of attack on any plant depends on the susceptibility of the host, the distance of original infection from the crown of the host, and the rate of growth up the root. The latter, in turn, is dependent on the pathogenicity of the strain and on environmental conditions.

The rate of growth of the fungus is of primary importance in the parasitic phase and its ability to survive is of major importance in the saprophytic stage; the environmental conditions favouring growth and saprophytic survival differ, and so, as far as possible, the subjects will be treated separately.

The problem of in vivo studies on the growth of G. graminis is that a change in one factor in the environment usually results in

an adjustment of all inter-related factors and it is very difficult to find many clear-cut reactions of take-all with individual factors in its environment. However, Garrett (1963) outlined conditions most favourable to the incidence and spread of take-all within a crop: the worst-affected areas of South Australia were on light sandy soils of alkaline reaction and farmers found that any cultivation which increased soil aeration further favoured the disease. In experiments, he found that G. graminis grows along wheat roots best in sand and that its growth in soil is favoured by any condition tending to promote better aeration, by a rise in the pH of the soil and by previous steaming, except in the case of more acid soils. He attributed the effect of steam treatment to some resulting physical or chemical change rather than partial sterilisation, because treatment with other partial sterilants did not encourage growth. Garrett investigated all these factors and later(1944b) succinctly ascribed the control of rate of growth of runner hyphae in poorly aerated or acid soils to accumulation of carbon dioxide at the root surface and an associated reduction in partial pressure of oxygen. He argued that in well-aerated soils carbon dioxide must diffuse rapidly from the root zone, and in alkaline soils a proportion is transformed into bicarbonate; light-textured soils also aggravate take-all attack because they are usually poor in nutrients and thus the host has less ability to survive by producing new roots.

Glynne (1935) reported little or no take-all on plots treated with ammonium sulphate, minerals or lime, or those with a pH less than 5.0. Although the incidence of G. graminis was very variable at all pHs, it tended to increase with increasing pH in wheat, but not in barley.

There are few other reports on the effect of soil pH alone, but the findings of Garrett (1944b) and Glynne (1935) accord with those of Ward and Henry (1961) working with liquid culture media, and the observation of Webb and Fellows (1926) that the relationship with pH varies with soil type and fertility.

Garrett (1939) did not investigate soil moisture per se but commented on its effect on the ability of the crop to withstand infection. McKinney and Davis (1925) explained this point more fully when they observed that although some plants appeared to be robust they were found to be severely attacked; symptoms only really showed during drier periods which were not necessarily associated with the best temperature for the parasite.

Åkerman, Granhall and Hagander (1935) stated that take-all was virtually absent during exceptionally dry seasons and virulent after heavy rain in May and June, while Škipsna reported from Latvia (1961) that the disease was widespread in wet years especially when rainfall was heavy during September and October, and April and May. Evidence for a positive correlation of disease expression with high rainfall is also given by Huber, Painter, McKay and Peterson (1968) who claimed that late autumn rains and moist soil early in spring was associated with epidemics because of enhanced nitrification. Warcup (1957) reported that the highest numbers of viable fungal spores and hyphae in a wheat field soil near Adelaide were found during the wet winter season and few were present in the dry summer period. Russell (1931), however found that both severity and incidence of take-all infection decreased with increasing moisture between 30% and 75% saturation in a pot experiment. This indicates that there may be a threshold value for moisture content above which aeration decreases to such an

extent that growth and incidence of G. graminis is adversely affected.

There are fewer opinions about the effect of temperature on take-all but Fellows (1941) stated that the best soil conditions for survival during winter were cool, compact and moist (60% W.H.C.) and Zogg (1963) that the elimination of G. graminis is associated with increasing temperature. Suzaki, Kasai, Nakaya, Araki and Takanashi (1957) claimed that the optimum field temperature for infection was 5 to 10°C but Garrett (1934) found that infection rose with temperature up to 24°C in a naturally sterile river sand and commented that, in general, antagonism is least pronounced in soils with low organic matter and low bacterial numbers, and most in evidence in fertile soils. It is generally known that microbial population levels and nitrogen transformation rates are usually higher in fertile and fertilised soils; the proximity of organic matter to the roots can affect root exudation and hence the microflora of the rhizosphere (Rovira and Ridge, 1973). Huber (1972) also found that temperature may affect the growth of the fungus through its effect on the soil microflora: in comparing the severity of take-all on wheat seedlings in sterilised and unsterilised soil at different temperatures it was found that seedlings in both soils reacted similarly at 13° and 18°C. However at 27°C most seedlings in sterile soil were killed whereas those in unsterilised soil were only slightly attacked. Henry concluded that the soil microflora had increased to such a degree at 27°C that its effect had been extremely antagonistic to take-all infection and growth.

Russell and Appleyard (1915) described how free air in soil to a depth of six inches was similar in composition to atmospheric air, except that the latter contained 0.03% CO_2 and 20.92% O_2 whereas soil air contained 0.25% CO_2 and 20.60% O_2 and

showed greater fluctuations in composition. They found that when the nitrate content of soil increased there was a decrease in oxygen, this effect being most marked in waterlogged soils.

Garrett (1936) formed a hypothesis that the growth rate of G. graminis along wheat roots is controlled by the partial pressure of undissociated CO_2 at the root surface: the spread of G. graminis is favoured at soil pH levels above 7 because of the lower ratio of undissociated CO_2 to bicarbonate ion. In 1937 he found that forced aeration of acid soils rendered them as favourable for G. graminis growth along wheat seedling roots as alkaline soils; he considered that growth in acid soils is retarded by accumulation of respiratory CO_2 and that alkaline soils probably accelerated growth by acting as a CO_2 acceptor in the root zone. However Winter (1940b) claimed that sterilisation which resulted in a decrease in respiratory CO_2, would only increase growth of runner hyphae in the very damp, dense clay soil-types with which Garrett worked. Nevertheless, Garrett in 1965 still affirmed that ectotrophic growth over the root system was largely related to the level of respiratory CO_2 at the root surface and claimed that the addition of organic manure helps to control take-all because it retards diffusion away from the root by raising the general concentration of CO_2 in the soil atmosphere.

Ferraz (1973) investigated Garrett's hypothesis by adding grassmeal to two contrasting soils and experimentally varying their pH. He confirmed that CO_2 retards growth of G. graminis but demonstrated that the effects of pH and aeration are separate when he found growth of G. graminis was not increased by removal of CO_2 from the unamended soils yet increased with

a rise in pH over the range pH 5 to 8. He concluded that the beneficial effects of soil alkalinity and of improvement in soil aeration on growth of G. graminis along roots are therefore probably unconnected and that the effect of aeration is most simply explained by an improvement in oxygen supply at the root surface.

Smith and Noble (1972) found that both var graminis and var tritici of G. graminis were tolerant to low partial pressures of O_2 and that growth was not restricted until below 0.2 atm, a level which they thought likely to occur only in waterlogged soil or at microsites. In contrast to this, they claimed G. graminis is unusually sensitive to CO_2 because, on agar, any increase above 0.0003 atm restricted growth; this is very low compared with Griffin's (1963) assertion that most fungi grow well up to 0.1 atm of CO_2 but it must be stressed that it is difficult to extrapolate from in vitro studies to the situation in the field. One important fact is that CO_2 is very soluble in water so that most gas dissolved in soil water and colloids is CO_2, with only a very little O_2, even though rainwater contains some dissolved oxygen (Russell and Appleyard, 1915). Fellows (1928) found hyphae of an inoculum placed below the surface of a liquid medium first came to the surface before further growth, and growth declined with reduction in oxygen from 21%, especially below 15%. Ferraz (1973) claims Garrett did not realise that in normal soils roots and their rhizoplane organisms are surrounded by a film of water when he interpreted his experimental results in 1936. Fellows' work suggests that not only does G. graminis have a growth requirement for O_2 but it also acts as a tropic factor.

When Winter (1940b) found that strong concentrations of aqueous extracts of heat-sterilised compost soil stimulated

runner hyphae development on wheat seedlings in sand, he attributed it to a change in nutrient content caused by the destruction of microbes.

Weste (1972) showed that G. graminis required an external supply of nitrogen for ascospore germination, growth and penetration, but when nitrogen and other nutrients are applied to the soil, G. graminis has to compete for nitrogen with plant roots and other members of the soil population. Vrany (1972) circumvented this competition by adding a urea solution directly to the leaves of wheat plants growing in inoculated soil in pots and found that fewer fungi grew on the roots. This resulted in an increase in mycolytic bacteria probably because of the existence of vulnerable inoculum unable to colonise the wheat host.

Most experiments on the effect of nutrients on root diseases deal with additions to the soil as in normal practice: Obst and Diercks (1972) reporting on agronomical trends in Germany, claimed that intensive green manuring and fertilisation with calcium cyanide tended to reduce G. graminis infection. Stetter (1971) found that phosphorus, potassium and nitrogen at 124 kg/ha in the form of calcium nitrate increased G. graminis in winter wheat, but nitrogen at 70 kg/ha decreased G. graminis in barley compared with unamended soils. Amelung, Dermoumi, Heide and Seidel (1971) added much larger doses of nitrogen in the form of cattle slurry: rates up to 200 kg/ha either reduced infection in wheat or caused no significant increase, but higher doses, particularly greater than 400 kg/ha "considerably increased the disease". These experiments imply that large doses weaken the resistance of the host or alter the soil ecosystem to the benefit of the parasite. The disparity between the threshold values above which the fungus

is benefited is probably caused by the different inherent fertilities of the soils.

Glynne (1935) noted that there was little or no take-all on plots with ammonium sulphate, minerals or lime, compared with untreated plots, and Garrett (1941) compared addition of full nutrients versus combinations of nitrogen, phosphorus and potassium in a pot experiment: phosphorus deficiency resulted in the most intense root infection in wheat; his overall conclusions were, however, that any nutrient deficiency, if sufficiently severe, is likely to increase loss of yield by disease. Garrett (1948) later ascribed the beneficial effect of adequate nutrients on take-all infection to the host's ability, in these favourable conditions, to produce crown roots faster than G.graminis can destroy them, but that too much nitrogen may increase the susceptibility of the roots to a greater extent than it helps disease 'escape'.

Hansen (1968) observed that ploughing-in legumes or adding 31 kg N/ha in the form of calcium nitrate increased yield of cereals but neither treatment reduced the actual incidence of take-all infection. Cunningham (1966b) found that nitrogen significantly reduced the number of diseased roots per gramme of total root but did not influence the number of infected roots per plant, that is, incidence of infection was unchanged, but nitrogen treatment increased the growth of uninfected roots, resulting in more resilient plants. The same reduction in severity but not incidence was noticed by Stumbo, Gainey and Clark (1942) with increasing rates of superphosphate. The nutrient deficiency most likely to aggravate infection depends on the soil: Garrett (1944b) declared that in Australia phosphate was most likely to be in short supply, whereas in England nitrogen

was more important.

In more recent papers the difference in effect between additions of ammonium-nitrogen and nitrate-nitrogen has been investigated: Huber, Painter, McKay and Peterson (1968) found that although increasing rates of ammonium sulphate and ammonium nitrate fertiliser reduced take-all severity in wheat, low rates of ammonium nitrate appeared to increase severity, although the percentage whiteheads decreased. They conclude that as there is a difference in response to form of nitrogen, root replacement is not the primary factor of disease control; instead they suggest the slower rate of nitrification of ammonium sulphate than ammonium nitrate results in more nitrogen being used by competitive and/or antagonistic micro-organisms, and thus, although G. graminis activity increased with increasing nitrogen, increased activity of other organisms, including, of course, the host, reduces take-all severity. However, Hornby and Goring (1972), working with a magnesium- and nitrate-deficient sandy loam, found that more wheat plants had take-all on plots without nitrogen than on plots with nitrogen additions, either as nitrate, or as ammonium-nitrogen with a nitrification inhibitor. Plants supplied with ammonium-nitrogen developed less take-all when magnesium was not deficient. In a comparison of nitrogen forms where there was no magnesium deficiency, take-all was shown to be least with a mixture of both forms of nitrogen, intermediate with nitrate-nitrogen alone and worst with ammonium-nitrogen alone, when the most extensive lesions on individual root axes occurred. They concluded that take-all attack is least when the amounts and ratio of ammonium- and nitrate-nitrogen are optimum for the growth of the host, and addition of ammonium-nitrogen favours G. graminis because it is

held on exchange sites in this form and is less mobile than nitrate-nitrogen in soils. Huber et al (1968) referred to the same property when explaining the opposite reaction of G. graminis to ammonium-nitrogen, although in Hornby and Goring's experiment the inhibition of nitrification of ammonium-nitrogen would probably adversely affect nitrifying organisms and therefore upset microbial balance in the soil. From this it is not clear whether G. graminis reacts positively to the form of nitrogen itself, or interacts with results of form of nitrogen on the soil and its ecosystem, which will obviously vary considerably with soil. Shipton (1972a) found no significant correlation between disease level and rate and form of nitrogen applied. Similarly Hornby and Brown (1977) could not determine whether there were any cause and effect relationships between ammonium:nitrate ratio changes in the rhizosphere and take-all.

Riley and Barber (1971) found that form of nitrogen influenced phosphorus uptake because of root-induced pH changes at the root surface of soyabean (Glycine max): adding ammonium-nitrogen decreased the pH of the rhizosphere and nitrate-nitrogen increased it. The change in pH was as large as 1.9 units in a soil of initial pH 5.2 and as small as 0.2 units in a soil originally 7.8. Addition of ammonium-nitrogen resulted in greater uptake of phosphorus, and the final phosphorus content of shoots and roots correlated with the pH of the rhizosphere but not of the bulk soil. They also record that soyabean root length decreased by 33% as rhizosphere pH increased from 4.7 to 7.5. Smiley and Cook (1971) related the interaction of form of nitrogen and take-all with changes in soil pH. They found that take-all control with ammonium sulphate in pots of fine sandy

loam (pH 5.5) failed when pH was raised to 7.7 by liming. However, control was obtained with no nitrogen or with calcium nitrate in silt loam, when it was acidified to pH 5.7; only ammonium sulphate was effective at its original pH of 7.5. Disease severity was not very highly correlated with bulk soil pH but more with rhizosphere pH: regardless of soil type, bulk soil pH, nitrogen fertility and form of nitrogen applied, severity declined as rhizosphere pH decreased below neutral. When soil was fumigated with methyl bromide severity did not vary above a rhizosphere pH of 5. They concluded that rhizosphere pH depended on the form of nitrogen added to the soil and below 5 it had a direct, chemical effect on severity; above rhizosphere pH 5 the effect was indirect being related not to the gases of the soil nor the chemical availability of nutrients but to the "activity of soil micro-organisms antagonistic to ectotrophic growth of G. graminis over the root surface". In 1973 Smiley and Cook claimed that disease severity decreased as rhizosphere pH decreased below 7.0 in unsterilised soil and was greatly reduced below 6.6. G. graminis did not grow in either sterile or unsterile soil below rhizosphere pH 5.0.

To summarise, any correction of nutrient deficiency normally benefits the plant more than G. graminis, and in some soils the reduction of rhizosphere pH with addition of ammonium-nitrogen may encourage certain micro-organisms which thrive on ammonium ions, making the nitrogen unavailable to the fungus until they die, when the nitrogen is released in the nitrate form.

These experiments imply an importance of rate of nitrification for the reduction of severity. Biederbeck and Campbell (1973) found that changes in temperature, such as the onset of the first cold spell each autumn and late frosts in spring

resulted in sudden flushes in nitrate-nitrogen production because of the death of micro-organisms unable to withstand the change in environmental conditions. Similarly, Huber et al (1968) claimed late autumn rains and moist soil conditions in spring enhanced nitrification and "were associated with take-all epidemics".

After harvest G. graminis survives saprophytically on infected root and stubble residues (Garrett, 1944b) and some weeds. Alternatively it may colonise grass weeds or pasture grasses and survive in an active state on these other hosts.

Although one-year rotational breaks of non-susceptible hosts are often considered adequate for control of G. graminis (Anon, 1970), in some conditions this has been found to be insufficient (Moore, 1948), because of the ability of the fungus to persist saprophytically. Conditions favourable for saprophytic survival are very different from those for parasitic activity: G. graminis is a parasite and cannot actively colonise dead host tissue; it is thus in even greater competition with other micro-organisms for external nutrients, especially nitrogen. Scott (1969b) observed that the relative importance of survival in root and straw tissue requires investigation; the degree of infection of straw varies considerably, but while there is always more infected tissue in roots, straw is less rapidly decomposed. Barley straw is not often visibly infected, but Chambers (1963) quotes W P Cass Smith that take-all survives longer on barley roots than on wheat roots because barley roots are coarser and therefore take longer to break down in the soil Although most observations about take-all survival have been made using buried straw, often inoculated with G. graminis some have investigated the infectivity of soil cores; results of these experiments are of more general application to

field conditions for both wheat and barley. Survival in the field may differ with soil and climate. In Australia MacNish and Dodman (1973c) found that the number of sites with stubble carrying viable G. graminis dropped from 90% in early February only to 82% in mid-November on a dry sandy loam of pH 8.5, but dropped from 90% in late January to 30% in late August during a warm, wet season on a dull-reddish-brown loam.

Garrett (1938) investigated the decline of viability of G. graminis in different soil conditions: he found it was "indefinitely postponed" in air-dry soil, in soil at 2^{o} to 3^{o}C, and under sterile conditions in a culture flask; it was less rapid in waterlogged soil than one maintained at medium moisture content. He concluded, therefore, that soil conditions least favourable for parasitic growth preserved it best during the saprophytic phase because they were also unfavourable for general soil microbiological activity. Fellows (1941) partially confirmed this when he compared the effects of different storage conditions on the survival of G. graminis in soil samples and found the best conditions were cool, compact and moist (3^{o} to 22^{o}C and 60% W H C), the most important factor being compactness. Garrett also found that addition of energy materials containing little or no nitrogen, such as glucose, starch and grassmeal increased loss of viability as this caused a flush in micro-organisms which competed vigorously for existing nitrogen. As rich, heavy soils support a larger population of micro-organisms the rate of decline of G. graminis was found to be greater than in poor, light soils. Garrett did not see any effect on decline of pH or moisture content of the soil over 20 to 30% saturation. Good aeration and fluctuating moisture in unglazed flower pots resulted in greater decline than in more uniform conditions and

was slowest in soil under uniform conditions in a closed incubator. Garrett stressed that decomposition of the infected straw did not necessarily coincide with decline of viability of the fungus. He suggested that the mycelium of G. graminis may perhaps even be a source of nitrogen for organisms decomposing the straw and therefore the rate of disappearance may be directly related to the quantity of nitrogen in the soil and in the straw. This is supported by the results of an experiment: additions of dried blood, containing 30% nitrogen, to the soil, delayed mycelial decomposition, but increased straw decomposition; additions of ryegrass meal resulted in delayed decomposition of the straw by causing a flush of saprophytic organisms which took up available nitrogen.

In a later paper Garrett (1940) considered the addition of nitrogen enabled the fungus to assimilate more of the carbohydrate of the straw, but where aeration allowed high microbial activity G. graminis disappeared faster because the straw was more rapidly consumed by all saprophytes, including G. graminis itself. Apparently, for the formation of fresh protoplasm one unit of nitrogen is required for about every fifteen units of carbon assimilated.

Garrett (1944a) buried infected wheat straw in washed quartz sand and found additions of 0.5 g N/100 g air-dry straw was optimum for survival, but additions of sodium phosphate did not significantly increase longevity. This suggested that nitrogen is required to form branch hyphae which can explore new areas of substrate, and that old mycelia die from carbohydrate starvation having exhausted the areas of substrate they occupy. This hypothesis is upheld by his finding that twice-weekly additions of 3% dextrose solution increased survival.

Garrett (1963) attempted to gain some idea of the cellulolytic ability of G. graminis compared with some other cereal root-rot fungi by incubating the fungi for seven weeks at 22.5°C on filter-paper cellulose. Pseudocercosporella herpotrichoides metabolised 0.8% of the dry weight of the filter paper and Helminthosporium sativum (Cochliobolus sativus) 14.8%; four isolates of G. graminis gave a mean loss of about 5.4% but varied from 2.5 to 8.4%. Although it does not necessarily correlate with competitive ability, low cellulolytic ability correlates with a high ability to survive saprophytically. This is one reason why it is generally considered that a three-year break from cereals is required to decrease incidence of P. herpotrichoides to a low level compared with only a one-year break under normal conditions for G. graminis. The principle was described in greater detail by Garrett (1966a): the extent to which rate of cellulose decomposition by a fungus was adequate to satisfy the requirements for saprophytic survival without a supply of nitrogen was expressed as a cellulolysis adequacy index. It was determined by dividing the percentage loss of dry weight of filter-paper, as found before, by the linear growth rate of mycelia in millimetres per 24 hours at 22.5°C; so that an index of less than one indicates that nitrogen is required for maximum survival. H. sativum was found to have the highest index (3.5) and additions of nitrogen decreased its survival in the soil. In a further experiment Garrett (1967) found the amount of nitrogen required for maximum longevity on filter-paper, and that any rate below this was insufficient for survival; thus the optimum is the minimum required for survival. In soil, 0.9g N/100 g soil was found to be too high for optimum

saprophytic survival.

Scott's (1969a) results of experiments with additions of nitrogen to the soil were similar to Garrett's (1944a: 1967), except that 0.1 g N/100 g soil, was supra-optimal for survival, presumably because the soil was naturally higher in nitrogen. Addition of 0.1 g N/100 g soil resulted in an equilibrium concentration of soluble nitrogen of 0.024 to 0.056 g/100 g soil. Despite Garrett's (1944a) finding that additions of 3% dextrose to flasks of infected straws increased survival, Scott found glucose at 2.5 g/100 g soil or 10 g/100 g straw increased microbial activity and shortened survival because of extra competition for nitrogen. However, Domsch and Gams (1972) asserted that a 10% concentration of glucose in agar inhibits growth of G.graminis in vitro, whereas dextrin had no such restriction. Scott confirms the finding of Lucas (1955) that pre-treatment of straws with dextrose decreased their infectivity when tested by Garrett's wheat-seedling test (1938).

Butler (1953b) investigated the infectivity of inoculated wheat straws by the same method after various lengths of time in jars of soil at room temperature and 45% saturation: only 3% of the straws were still infective after 28 weeks when kept in soil with a nitrogen content of 0.0005 g/100 g air-dry soil in contrast with 40% in soil containing 0.0222 g N/100 g. Later, Butler (1959) compared fertile and infertile soils and found survival was reduced to 50% after 5 months in the infertile, cultivated soil but only fell to that level after 8 months in the fertile ley soil which had 50% more organic carbon and twice the amount of nitrogen. After 40 weeks only 20% of the inoculated straws were viable and after 52 weeks no G.graminis could be

recovered. However, Lucas (1955) found a possible source of discrepancy in the wheat seedling test so that although figures are comparable within this experiment, absolute figures for survival do not necessarily bear any relation to possible survival in the field. Shipton (1972a) found inoculum could survive for up to 66 months after the last susceptible cereal crop. Petersen and Christensen (1968) recorded survival of G. graminis in buried straw for 3½ years, but its pathogenicity declined rapidly after one to two years. Slope, Henden and Etheridge (1969) stored 300 cm^3 soil cores from an infected wheat crop in "favourable conditions"; after two years several of them were still infective so they concluded that, in some instances, a 2-year break from cereals would be ineffective for take-all control. Glynne (1965) observed that take-all developed most quickly, even after a 2-year cleaning period, on land where there had previously been a severe attack. Lucas (1955) found that saprophytic colonisation of wheat straw could only occur "to a negligible extent" in the field when he used naturally infected stubble plants as inoculum, thus implying agreement with Butler (1953a) that G. graminis had "only a limited ability" to colonise wheat straw saprophytically in competition with the microflora of the soil.

Van der Watt (1965) investigated nutritional and possible microbiological differences between six different cereal soils and found they had little effect on the survival of G. graminis, although high applications of nitrogen shortened the persistence of G. graminis in all soils. Similarly, Chambers and Flentje (1969) observed that the viability of G. graminis decreased very slowly in unamended, irradiated sterile soil; contamination of the soil by other organisms accelerated loss of viability, but

this effect could be reversed if nitrogen were added. Survival appeared to them to be more dependent on the availability of nitrogen than on competition and antagonism from other micro-organisms. As nutrients are an essential factor in competition their conclusions may be more correctly stated that survival depended more on nutrition than on other factors of competition or antagonism by other micro-organisms.

Contrary to the findings of Van der Watt, Lester and Shipton (1967) reported considerable differences in the ability of soils to reduce the amount of pathogen surviving from added inoculum and implicated this as a factor in the phenomenon of take-all decline.

The ecology of G. graminis can be summarised by abstracting the most important points from the review of literature. G. graminis grows best when it is subjected to the least amount of competition and antagonism; it must have a susceptible host before it can actively increase its mycelial bulk and prefers warm and moist, but not wet, conditions because oxygen supply is very important. A pH of less than 5 in the region of the fungus seems to have a direct and detrimental effect on growth; above rhizosphere pH 5 the effect is indirect because of the activity of micro-organisms existing in particular soil conditions of which pH is an integral factor. Growth of G. graminis appears to be inhibited by excess carbon dioxide but the work of Ferraz (1973) implies that any gas which in excess is symptomatic of low oxygen concentration, for example, ethylene, will be negatively correlated with growth. G. graminis requires a supply of nitrogen for initial colonisation of its host; thereafter the host may provide all necessary nutrients.

G. graminis can survive in an active form on some plant species which are not actually susceptible, provided the fungus can absorb nutrients from the soil, but if no host or 'carrier' plant is available, it persists saprophytically on dead host material colonised during its parasitic phase. The ability of G. graminis to colonise 'clean' crop debris is thought to be very limited. Saprophytic survival is optimum when its metabolic rate is lowest. It thus requires very little oxygen and the minimum of nutrients: only enough for new mycelial growth to colonise fresh areas of substrate before starvation occurs. In contrast with these optimal growth conditions for the fungus, the conditions for optimum attack are changed by the microflora of the soil and by the host. So great are the effects of these organisms that, except for inadequate oxygen and pH lower than 5, the incidence and severity of attack of take-all is dependent, either directly or indirectly, on them. Therefore, any environmental condition or modification which benefits the host or soil microflora as a whole will decrease the importance of take-all disease to its host.

The effect of cultivations on take-all

The effects of cultivation on take-all have been investigated for many years and the findings generally confirm that any treatment which benefits the crop and encourages the soil microflora decreases the importance of take-all attack. Škipsna (1961) recommended cultural measures such as ploughing-in stubble, organic manuring, liming acid soils and drainage.

Conventional tillage is mould-board ploughing followed by discing or harrowing, and post-planting cultivation. Ploughing and other primary tillage operations, for example rotovating,

deep ploughing and subsoiling loosen the soil and increase porosity. Secondary tillage such as applying fertilisers, sowing, rolling and caring for the crop, controlling pests, harvesting the crop and disposing the residues repack the soil (Roo, 1969).

Chambers (1962b) reported that depth of cultivation had little effect on incidence of take-all: a significant decrease was found only in one soil when comparing 4 versus 8 inches (10 versus 20 cm) depth. A rigid-tine scarifier, similar to a chisel-plough, did not significantly reduce incidence compared with ploughing, but ploughing resulted in better weed control. Scott (1969b) found that G.graminis survived longer on artificially inoculated wheat straws when they were left on the surface of the soil than when buried, and for very much longer when removed from contact with the soil. He claimed that early post-harvest rotovation significantly reduced disease incidence, probably because of enhanced microbial activity and competition for nitrogen in the well-aerated compost of soil and stubble. Cunningham (1967a) reported that incidence of severe take-all infection was greatest in a spring wheat crop after ploughing at 4 inches and was less after ploughing at 8 inches than at 12 inches (30 cm). As the 12 inches ploughing failed to invert the thicker sod completely, he also concluded that incidence was directly related to the amount of surface-stubble debris. Deep ploughing decreased surface inoculum and the amounts of severe infection and although it increased plant growth this resulted in conditions more favourable for light infections. Deep ploughing may bury infectious material below the main root zone and decrease injury, as Fellows and Ficke (1943) found that serious injury is most likely to occur

when inoculum was placed 3 inches (7.5 cm) or less from the seed. Chambers and Flentje (1968) found saprophytic survival was better at 1 inch (2.5 cm) than at 6 inch (15 cm) depth in the field, but Glynne (1965) observed that deeply-buried inoculum could survive to form a "reservoir of infective material". Warcup (1957) reported that, in general, there was little difference in fungal counts between 1 inch and 3 inch deep samples, but significantly fewer fungal units were isolated from a 6 inch horizon. This could mean less favourable physical and chemical conditions for saprophytic survival of G. graminis, although it is not very sensitive to oxygen level during this stage: it could also result in a lowered activity of competitive organisms in the rotting material and the overall effect may increase persistence. Ogilvie and Thorpe (1962) observed that certain organisms, shown to be antagonistic in vitro, such as Actinomycetes and Penicillium species, are prevalent in rotting material.

Sewell and Melchers (1924) claimed that September ploughing, with or without discing, was detrimental to the development of the disease, but Endeladze (1968) found the greatest incidence of take-all when ploughing at 10 inches (25 cm) depth immediately after harvest was followed by harrowing. Cunningham (1967a) recorded greater grain yields after ploughing in February than in early April probably because it produced a better seed bed; the effect on disease incidence and severity was small. In the Netherlands, Van de Laar (1931) reported that G. graminis was more prevalent on early sown winter wheat (24th October) than on wheat sown later (10th December) and Van der Watt (1965) advised that after the first rains in the

"winter-rainfall region of the Cape" a delay in sowing "avoids the consequent disease increase". Bockmann (1934) also advised late sowing. Cunningham (1966b) observed that the incidence of take-all decreased with earliness of ploughing and lateness of sowing, and was increased as the interval between the two was shortened; nevertheless, when nitrogen was adequate the adverse effect of late sowing on yields was greater than the advantages of controlling disease. Rosser and Chadburn (1968) recorded that in the very severe winter of 1962-1963, winter wheat crops sown later than mid-October were more heavily infected with take-all than those sown earlier. This was probably caused by the severe winter extending the most vulnerable growth stages in the host. Shipton (1972a) attempted to reduce the amount of inoculum surviving to infect a subsequent crop by burning wheat and barley stubbles after harvest with a high output flame cultivator, but found no reduction in take-all and eyespot incidence. This confirmed the findings of Slope, Etheridge and Callwood (1970).

Doling and Batts (1960) found that the incidence of take-all doubled when the sowing rate of the winter wheat variety, Bersee, was increased from 1½ to 2½ bushels/ac (1.35 to 2.25 m^3/ha) in the fourth consecutive wheat crop, but rate of sowing had no significant effect on incidence when winter wheat was grown in rotation with other crops.

G. graminis thrives in well-aerated soils; so Garrett (1937b) recommended shallow cultivations and repeated rolling to give a firm seed bed if soil is light. The decrease in aeration must not be excessive, however, as Vomocil and Flocker (1961) claimed that plant growth and yield decreases appreciably below

soil air space (porosity) of about 10 to 15% of the total soil volume. Winter (1939) also reported reduced incidence of take-all after compaction of loose soils but later explained that consolidation is only effective if the inoculum is far enough away from the stem base to let the host establish itself (Winter, 1940a). Consolidation of light soil may benefit the crop by affecting soil parameters such as nutrient availability. Passioura and Leeper (1963) reported that consolidation relieved manganese deficiency in light soils: they suggested that this was because of the greater area of contact between roots and solution as the result of the production of thicker roots in compacted soils. Buckman and Brady (1969), however, thought manganese availability is related more to soil aeration.

The recent revival of interest in soil structure and cultivations occasioned by the Agricultural Advisory Report (1970), has led to the investigation of the agronomic effects of minimal cultivations. Brooks (1967) found that direct drilling of winter wheat into paraquat-killed grassland or stubble reduced take-all. He thought that the effect was more likely to have been caused by biological factors limiting the spread of take-all than the fungicidal action of paraquat. G.graminis spread more rapidly along wheat roots in soil from ploughed plots than from direct-drilled plots. As the top two inches of unploughed soil produced more carbon dioxide than at any level in the ploughed soil, the former appeared to support a larger microflora and Brooks felt that this limited the growth of the pathogen. Brooks and Dawson (1968) stressed that incidence of infection by take-all was less under direct drilling, in spite of heavy couch infestation; although fewer plants were infected they were

more severely damaged, perhaps because the roots were in closer contact with the infected residues of the previous crop. They found that the inoculum was more widely distributed in the ploughed than in the direct drilled plots where the soil had not been disturbed, but warned that the drilling slits might permit the fungus to spread very rapidly despite minimum tillage.

The effect of manuring on take-all

Garrett (1944a) suggested growing catch crops to starve the fungus during its saprophytic stage. Similarly Garrett and Mann (1948) and Hansen (1968) discussed the use of undersown trefoil (Medicago lupulina) as in the Chamberlain system of continuous spring-cereal growing. After harvest the autumn growth of the legume appeared to assist in starving out the fungus by competing for residual nitrogen in the soil. However Hansen found that although this increased yield of the subsequent crop, and therefore decreased severity, incidence of take-all did not decrease. Scott (1969b) found that in a glasshouse experiment survival in buried winter wheat straws was slightly more prolonged in fallow soil than under clover, but undersowing red clover in a field trial did not reduce the incidence of take-all, probably because of above-ground survival of G. graminis in unploughed straw. Shipton (1972a) applied nitrogen to barley and wheat stubble after harvest to encourage other saprophytic organisms, but incidence of take-all was not reduced.

Obst and Diercks (1972) observed that intensive green manuring and fertilisation with calcium cyanamide helped to reduce take-all 'infection', but Åkerman, Granhall and Hagander (1935) reported that G. graminis was stimulated by stable manure which was not completely rotted; this probably occurred because the

saprophytic microflora would have been occupied well into the growing season and not liberating nutrients from the manure as they would have been if it had been added to the soil in a more degraded state.

Salt (1959) found that applying 3 or 6 cwt/acre (377 or 753 kg/ha) Nitrochalk to winter wheat in April resulted in more yield and less take-all and eyespot than fertiliser applied in March. Fertiliser applied in May increased weeds, decreased yield and did not decrease take-all. Huber (1972) recorded that take-all was highest in spring wheat when nitrogen had been applied the previous autumn. Yield could be brought up to the level of that of an unfertilised crop if another application of nitrogen is given in the spring, but take-all was lowest when nitrogen was added only in the spring. Cunningham (1966a) could find no influence of time of fertiliser application on take-all. Shipton (1972b) stated that in a spring cereal monoculture system nitrogen did not influence take-all until the point of maximum incidence and severity, when it caused a reduction in disease level in addition to that associated with take-all decline.

The effect on take-all of chemicals applied to a cereal crop

Other methods of combatting take-all which have been investigated include the use of herbicides and pesticides. Cunningham (1966a) found that the herbicides MCPA and 2,4-DP decreased the incidence of take-all only when nitrogen was broadcast, not when it was drilled, because broadcasting results in the weeds receiving more nutrients than if the fertiliser is drilled with the seed. Wilkinson and Lucas (1969a) thought

herbicides may alter the ability of saprophytic fungi to colonise plant tissues by selective fungicidal action which, in turn, may affect the rate at which these substrates are subsequently decomposed. Although Brooks (1967) concluded that paraquat did not seem to have any fungicidal properties, Wilkinson and Lucas (1969b) reported that paraquat depressed growth of G graminis and Trichoderma viride. Nilsson (1973c) observed that treatment of spring wheat with 1% to 10^{-7}% solutions of mecoprop resulted in roots or root tips becoming bulb-like and the tissues rather 'loose' so that fungal penetration was easier than normal. The herbicide therefore increased infection although the hyphae of G graminis were abnormal at concentrations of 10^{-5}% and more. Nilsson (1973d) tested the herbicides tribunil, sinadal, basanor and oxytril 4 in a field trial. Grain yield was slightly greater than the control with all herbicides except oxytril which significantly increased the incidence of take-all and eyespot. Basanor increased take-all infection very slightly.

Slope and Last (1963) reported decreases in take-all severity in wheat grown in pots when the pesticides aldrin, dieldrin, chlordane and heptachlor were mixed with soil. Heptachlor had the greatest effect: 20 kg/ha of active ingredient decreased the proportion of infected seminal roots from 96 to 37% and that of infected crown roots from 66 to 2%. At this rate thiram did not affect the disease. Williams (1969) found that nabam had no effects on G graminis, but formalin usually controlled the fungus in the year it was applied. Ebbels (1969) observed that dazomet affected take-all in much the same way as formalin: after the initial decrease, incidence increased in the second

cereal crop. The effect of a second application is less than the first and at Rothamsted take-all seemed as prevalent on plots treated with dazomet every year as on rotovated and unfumigated plots. He thought that the increase in the second crop after fumigation with D-D meant that the populations of G. graminis recover sooner from fumigation than other soil organisms which keep the fungus in check. Hornby (1969a) found that MBOA did not affect the ability of G. graminis to infect wheat roots; methyl bromide, chloropicrin, D-D mixture, dazomet and formalin decreased take-all incidence, but mercury salts did not (Williams and Salt, 1970).

Cunningham (1967) and Gerlagh (1968) observed that CCC, an inhibitor of shoot elongation in wheat and oats, increased the percentage of whiteheads in a crop.

There are reports of reductions in take-all by the use of carbendazin (Scott and Hollins, 1976, 1977; Benada, 1976) and benomyl (Grisenko and Pariĭ, 1977; Benada, 1976).

Biological Control

Škipsna (1961) advocated sowing high quality seed of resistant varieties treated with spores of cultured T. viride and trichodermin, as measures against take-all. Ponomareva (1965) found that seed treatment with a spore suspension was less effective than soaking in trichodermin-3. Brants (1971) reported that Pythium sylvaticum could be infected with the deleterious virus, tobacco mosaic virus, in vitro. Infecting G. graminis with virus-like particles, even if they are shown unquestionably to reduce pathogenicity of the fungus, would probably not be feasible. However encouraging populations of Phialophora radicicola var graminicola (PRG), as suggested by Deacon (1973a),

might control G. graminis in a manner similar to T. viride in citrus groves where its occurrence is negatively correlated with Fusarium solani (Joffe, 1966), the fungus causing damping-off of citrus seedlings. Gutteridge and Slope (1978) decided that evidence suggesting that PRG may contribute to the decline of take-all in continuous wheat was inconclusive.

Work in France has suggested a possible use of hypo-aggresive strains of G. graminis as a form of biological control (Lemaire, Jouan, Coppenet, Perraton and Lecorre, 1976).

Use of resistant varieties

Škipsna (1961) recommended the use of resistant varieties of wheat, but Åkerman et al (1935) could find no well-marked differences among wheat varieties and the Agricultural Gazette of New South Wales (1962) stated that there were no resistant varieties. However, Gorska-Poczopko (1963) reported that two varieties of wheat seemed more susceptible than another two, and differences in degree of infection and yield response between winter wheat varieties have been investigated by Scott and Hollins (1976, 1977).

Nilsson (1969) recorded differences in varietal resistance in both wheat and barley. By using specially controlled culture conditions, Nilsson (1973 a and b) found substantial differences in resistance between eight winter wheat varieties. As the differences were more pronounced when the host was inoculated with G. graminis at 22 or 30 days old than at 8 or 15 days old, the resistance appeared to be concerned with tolerance of the disease. All varieties were heavily infected when inoculated at an early age. It is difficult to forecast whether a variety will be found that is as resistant to take-all

as some wheat varieties are to eyespot, but it is pertinent to attempt to analyse why different graminaceous species vary in their susceptibility to G. graminis and why, although runner hyphae may be found on dicotyledons, the fungus never invades the tissues. Jensen and Jørgensen (1973) tested the relative tolerance of wheat, barley, octoploid Triticale, rye and oats under controlled conditions. Oats were highly resistant. Wheat suffered greatest grain yield loss and although rye was moderately resistant, under severe conditions its yield was halved. Barley and Triticale reacted intermediately between wheat and rye. Linde-Laursen, Jensen and Jørgensen (1973) investigated the susceptibility of some graminaceous species; they concluded that high levels of resistance must be rare within Triticeae and that where resistance was found, for example in Haynaldia villosa, it cannot be exploited successfully in breeding wheat for resistance to G. graminis because of the dilution of the resistance when combined with Triticum germplasm.

Oats are resistant to G. graminis var tritici because they produce an antifungal substance toxic to the common form of G. graminis. G. graminis var avenae, however, can attack oats because it produces a specific enzyme, avenacinase capable of destroying the toxin (Turner, 1961). Defosse (1966) found that when root extracts of oats, and also maize and clover, were incorporated into agar, growth of G. graminis (var tritici) was completely inhibited. Barley produces antifungal compounds (Ludwig, Spencer and Unwin, 1960; Koshimizu, Spencer and Stoessl 1963), but none have yet been shown to be toxic to G. graminis.

Conclusion

Take-all is rarely a problem in cereal production where

rotations between susceptible or non-susceptible crops are adequate. G.graminis has a limited power of saprophytic survival and is reduced to negligible proportions with breaks of one or two years of non-susceptible crops. Economic pressures, however, have forced farmers to increase the proportion of cereal crops on their farms with the result that rotational practices have suffered. This has led not only to problems of severe outbreaks of foot and root rots but also to epidemics of foliar diseases. In some areas farmers have completely abandoned rotations, but yields have not followed the disastrous pattern which might have been theoretically forecast. This may be partly attributed to the introduction of methods for controlling foliar diseases and partly to the phenomenon of take-all decline which limits the effects of take-all in continuous cereal growing. Although the mechanism of TAD is not fully understood the pattern of its development suggests that, provided high standards of management are maintained, continuous cereal growing may have advantages over rotations with only occasional break crops, when the disease attack is at its peak.

Where cereal crops are grown in succession, early ploughing of the stubble has been practised to encourage the decomposition of infected debris and thereby reduce the level of carry-over of take-all. However, it was found that the introduction of direct drilling techniques with minimal cultivations results in some control of take-all, which is related to the distribution of organic matter, microbiological activity and lack of soil aeration. A low oxygen level restricts the spread of take-all within the crop and as long as root growth is not impaired can be responsible for a reduction in take-all,

hence cultivations, such as rolling, which compact the soil are recommended for soils which are naturally well-aerated.

The control of foliar diseases is thought to result in greater yield responses than expected because destruction of leaf tissue reduces root growth and therefore the ability of the crop to withstand root attack. Similarly, any other treatment which improves root growth, such as the correction of poor drainage and low soil fertility, will reduce losses caused by take-all. Any factors which encourage vigorous crop growth will tend to alleviate the adverse effects of take-all. Often the 'best' agronomic practices enhance the growth of G. graminis but enable the crop to withstand the disease better; the particular cultivations and manuring which will decrease the effect of take-all in a crop will vary with soil conditions. Fertiliser applications generally decrease the effect of take-all but ought to be applied according to the needs of the crop; a higher nitrogen rate is usually required with minimal cultivation systems. Organic and 'green' manures decrease take-all damage only if they rot down in time to benefit the next crop nutritionally.

The use of undersown clover or grass and clover in the Chamberlain system as an alternative to the yearly cereal/fallow 'rotation' of the spring cereal system has been practised in the past and may have further application in the future.

The possibilities of biological control have been examined but there has, as yet, been no general practical application of this approach. Some of the most recent studies in this area have indicated the possible benefits of the use of

grass leys to establish, either naturally or by inoculation, populations of Phialophora radicicola, an avirulent parasite which competes with G. graminis.

No chemical has been developed for use against G. graminis but any chemical which depletes populations of weeds should benefit the crop. Obviously the few chemicals which have been found to increase take-all infection, such as oxytril and basanor, ought to be avoided. It was found that G. graminis can tolerate the repeated use of some fungicides and nematocides, for example dazomet or formalin. The possibility of exploitation of any specific chemical method of control seems limited on economic grounds.

The development of take-all resistant varieties of wheat and barley seems unlikely in the near future, as resistance genes have only been found in related species of grasses. Perhaps, in the long term, genetical resistance may be introduced into commercial varieties, although the variation in pathogenicity, already evident in the species G. graminis, may be a complicating factor.

CHAPTER 1

SOUTH ROAD LONG-TERM BARLEY EXPERIMENT

A continuous barley trial at Langhill Farm, Bush Estate, Midlothian has been the subject of a PhD thesis (Lockhart, 1972) and a paper by Holmes (1976). The take-all results from 1968 to 1974 were given and briefly discussed by Lockhart, Heppel and Holmes in 1975; however, further developments in the trial and in recent research make a full discussion at this stage not only of scientific interest but also of relevance to cereal agronomy and practice. The present work includes later findings from this trial and discusses the results of the experiment in the light of current research information.

Materials and method

In October, 1965, a 1.94 hectare site was established on South Road Field for a continuous barley experiment. A three-year ley from 1961 to 1964 was followed by potatoes in 1965. The trial has been sown every year since 1966 with the spring barley variety, Zephyr; the first two years were occupied with the establishment of uniformity. Applications of slag and sulphate of potash in December 1966 were ploughed down to a furrow depth of 25 cm.

From October, 1967, eight replicates of four cultivation treatments were imposed, each being divided into four subplots for nitrogen treatments. The main plot treatments were: deep ploughing, shallow ploughing, chisel ploughing, and direct drilling with minimal cultivation; the subplot treatments were: no applied nitrogen, 50 kg N/ha, 100 kg N/ha, and 150 kg N/ha. The area of each mainplot was 49.4 x 12.2 m; this was divided into subplots of 24.7 x 6.1 m. The plot layout is illustrated in Appendix I.

Deep ploughing penetrated to about 30 cm and shallow ploughing to about 15 cm. The depth of the three chisel ploughings was the maximum possible in the conditions: between 25 cm and 30 cm. Nitrogen in the form of ammonium nitrate (25% N) was applied by hand after drilling. A triple disc Fernhurst drill was used for all plots, but had extra weights fitted for direct drilling the minimal cultivation treatment plots. Within each plot a central area was reserved for grain yield measurements; all other variates were measured from samples taken from the two outer areas of the plot.

Samples for the measurement of take-all were taken twice during each growing season: late June or early July and the second week in August. In 1977 the trial was in its twelfth consecutive barley crop and its tenth year under the different cultivation and nitrogen treatments. The early sampling estimated the incidence of take-all throughout the crop at growth stage 5 on the Feekes Scale (see Large, 1954) and the later, the incidence at growth stage 10.5 to 11.00. Normally the incidence assessments are combined with estimates of severity which, at the early sampling, provide a measure of the inoculum potential within the top 7.5 to 10 cm of soil.

Since 1968 the method used for assessment of take-all incidence was that suggested by D B Slope (personal communication to D A S Lockhart, 1968). He considered that a reliable estimate of incidence can be obtained from lifting five samples of 30 cm lengths of drill at pre-determined, randomly-distributed positions per plot. After the plants had been carefully dug up with a hand-fork to preserve as much root as possible, each 30 cm subsample

was shaken to remove loosely adhering soil and rolled in a narrow canvas strip to keep it separate from the other subsamples removed from the plot. In this way the exact original location of the subsample within the plot could always be traced.

The samples were then soaked in water and the number of plants and tillers in each subsample were counted. When the roots had been finally washed free of any soil they were examined under water in a white tray. The number of plants in each subsample bearing 'typical' take-all symptoms of black roots or black lesions or mottles on their roots was counted and the figure divided by the number of plants in the subsample to give the percentage plants infected.

Severity of infection was assessed at the early sampling date by dividing the number of infected roots by the number of infected plants per subsample. Initially severity was assessed at the later date by scoring infected plants as follows:

No obvious visible infection	0
Seminals and 1 or 2 crown roots infected	1
Crown roots moderately infected	2
All roots totally infected	3

The scores were then totalled and divided by the total number of infected plants in the subsample. From August, 1973, however, severity was measured in August using the following scheme as it was felt that it might be more sensitive to differences in crown root infection between treatments:

No visible infection	0
Seminals infected, but no crown roots	1
One or 2 crown roots infected (and usually seminals)	2
All crown roots (and usually seminals) infected	3

The author assisted at take-all assessments carried out by D A S Lockhart from 1968 to 1970 and was subsequently responsible for those in 1971 and 1973 to 1977; none were made in 1972.

When the crop was ripe the centre of each plot was cut by a six-foot combine-harvester fitted with a special weighing hopper. The yields were corrected to 15% moisture content.

The results of take-all incidence and grain yields were subjected to statistical analysis to compare not only the effects of cultivation and nitrogen treatments, but also possible differences between replicates 1 to 4 (Block I) and replicates 5 to 8 (Block II). The soil type in Block I is predominantly Macmerry, a sandy loam. The soil in Block II is approximately half Macmerry and half Winton, a sandy clay loam; see Appendix I.

Results

1) Incidence

The patterns of incidence of take-all at the early (June) and late (August) assessments from 1968 to 1977 are given in figure 1. The effect of cultivation treatments are shown in figures 2 and 3 and of nitrogen treatments in figures 4 and 5.

The average infection level throughout the crop was low until August in the 9th year when it reached 35%. Thereafter 41% of the plants were infected in late June of the 10th and 11th seasons and 25% in the 12th. Incidence in August reached a peak of over 80% in the 11th year and subsequently decreased to 44%.

The same general trend over the years is seen when each cultivation and nitrogen treatment is considered in turn at both samplings.

At the earlier sampling in the 4th and 5th years there

Fig 1

The development of take-all in the South Road trial from 1968 to 1977 at early (I) and late (II) assessments

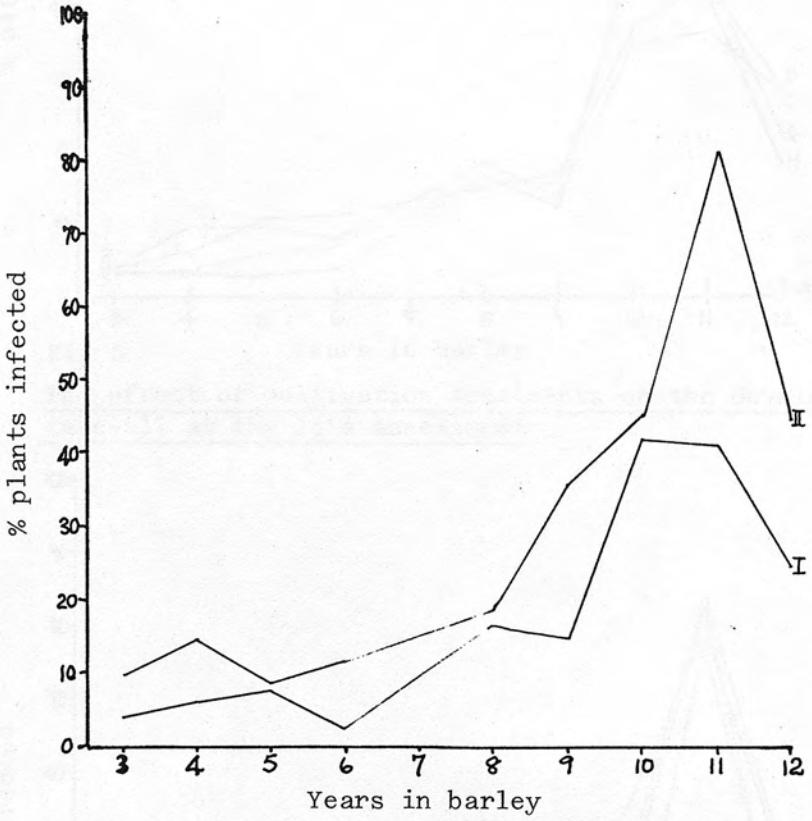


Fig 2

The effect of cultivation treatments on the development of take-all at the early assessment

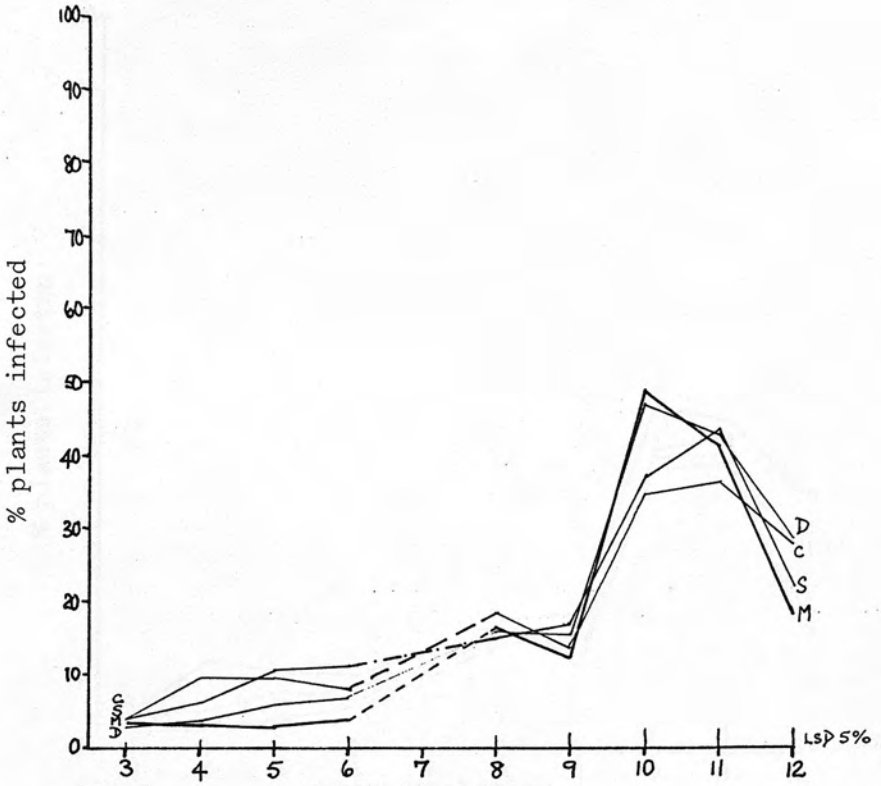


Fig 3

The effect of cultivation treatments on the development of take-all at the late assessment

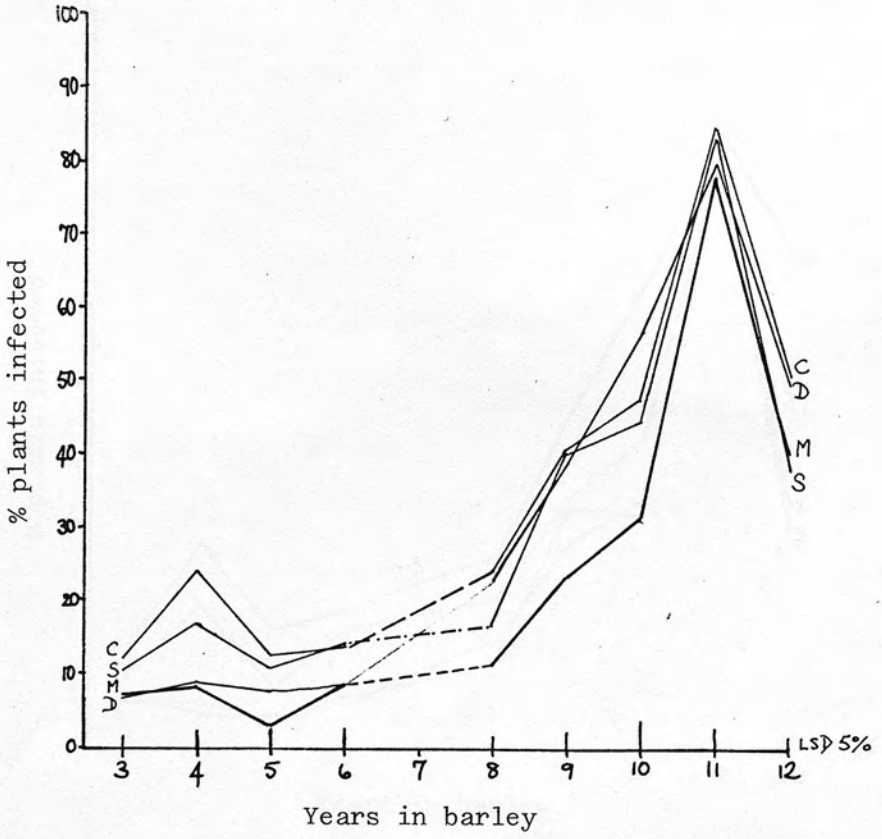


Fig 4

The effect of nitrogen treatments on the development of take-all at the early assessment

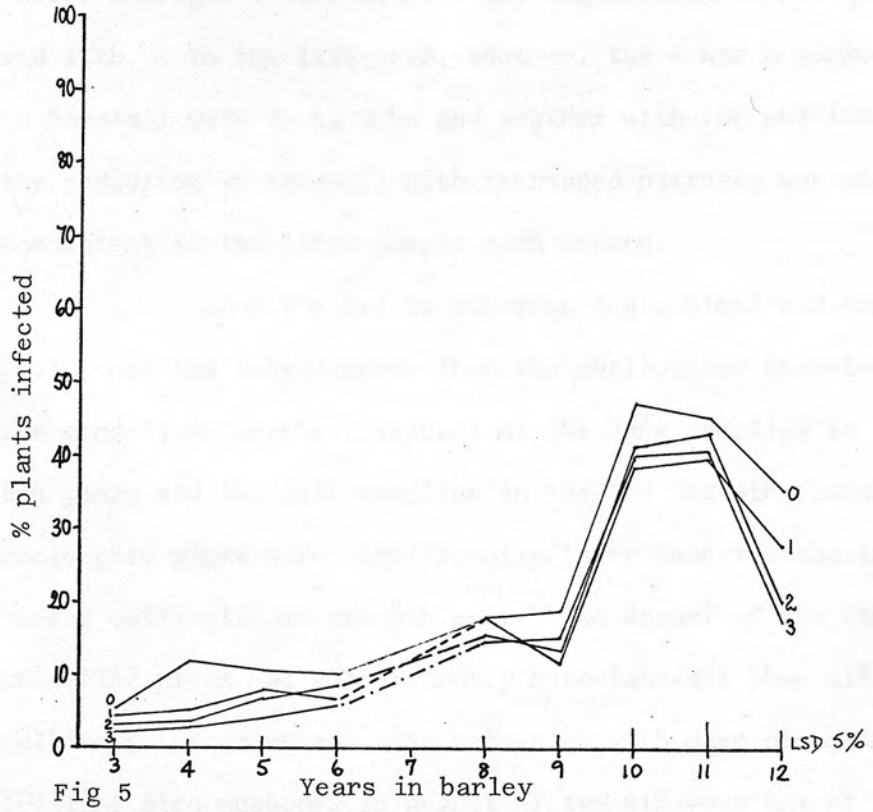
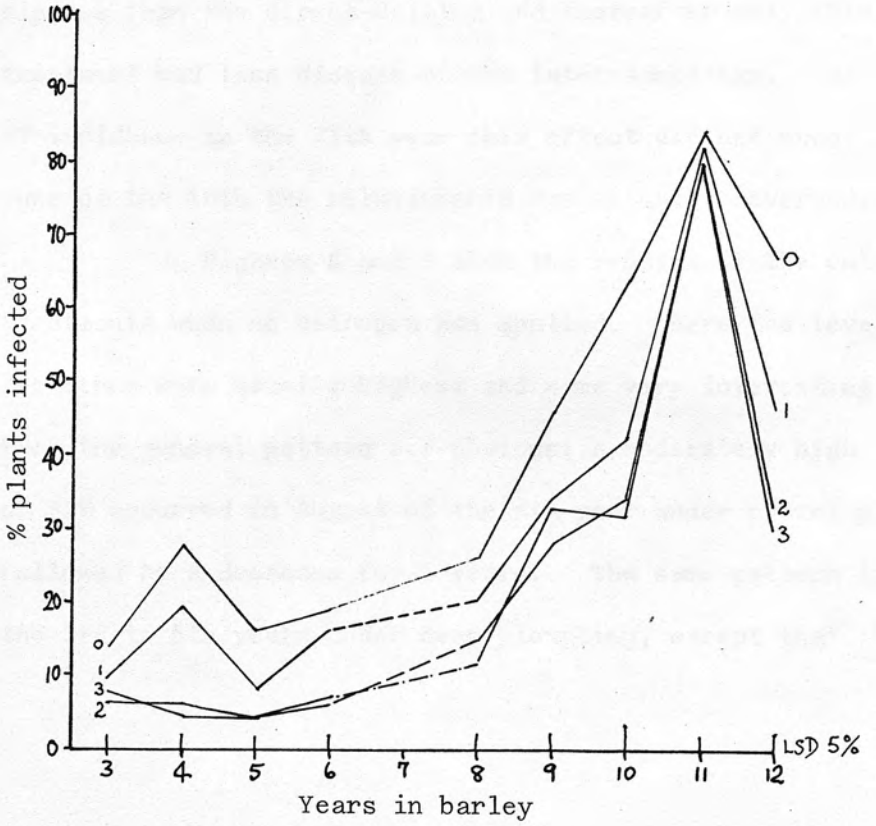


Fig 5

The effect of nitrogen treatments on the development of take-all at the late assessment



was a clear decrease of incidence with increasing nitrogen but in the middle and later years the response was less uniform and, indeed nitrogen ceased to have any significant effect in the 10th and 11th. In the 12th year, however, there was a marked decrease in take-all with 50 kg N/ha and another with 100 and 150 kg N/ha; the reduction in take-all with increased nitrogen was more consistent in the later sample each season.

From the 3rd to 5th crop the minimal and deep cultivation treatments had less disease than the shallow and chisel-ploughed. The reductions were significant at the June sampling in the 4th and 5th years and the late sampling in the 3rd and 4th years; only the unploughed plots were significantly lower than the shallow and chisel cultivated in the 5th year. In August of the 4th year the chiselled plots had significantly more take-all than all other cultivated treatments. The reduction with deep ploughing and direct drilling also occurred in August of the 6th year but at the earlier sampling all three cultivated treatments had significantly more disease than the direct-drilled and thereafter only this later treatment had less disease at the later samplings. At the peak of incidence in the 11th year this effect did not occur and in June of the 10th the relationship was actually reversed.

Figures 6 and 7 show the results of the cultivation treatments when no nitrogen was applied. Here the levels of incidence were usually highest and some very interesting deviations from the general pattern are obvious: a moderately high infection of 52% occurred in August of the 4th year under chisel ploughing followed by a decrease for 2 years. The same pattern is seen in the 3rd to 6th years under deep ploughing, except that the peak of

Fig 6

The effect of cultivation treatments without addition of nitrogen fertiliser on the development of take-all at the early assessment

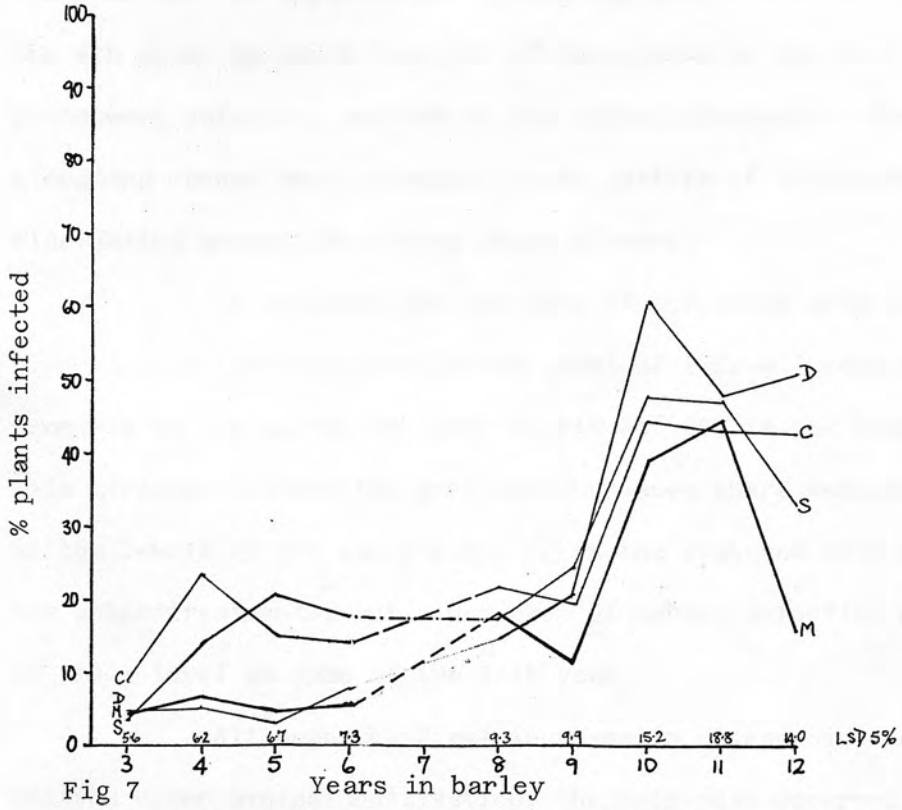
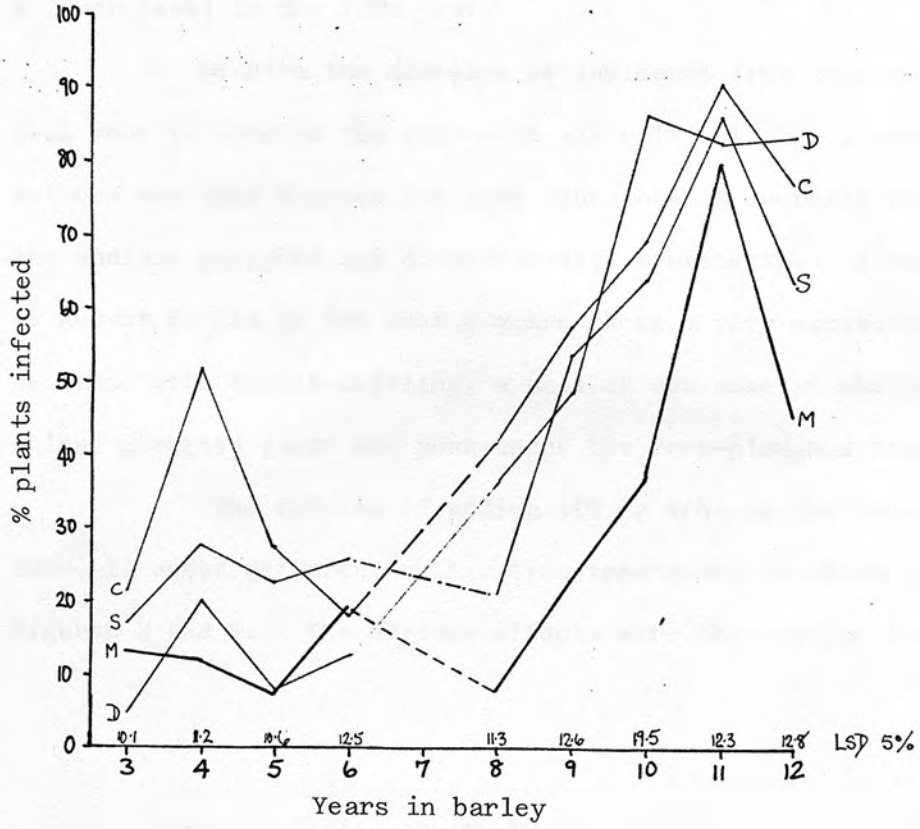


Fig 7

The effect of cultivation treatments without addition of nitrogen fertiliser on the development of take-all at the late assessment



infection was lower (20%). This rise and fall also occurred in the direct-drilled plots with the peak of 19% delayed until the 6th year and the apparent subsequent depression continuing into the 8th year, by which time 37% of the plants in the deep ploughed plots were infected, and 41% in the chisel ploughed. Shallow ploughing caused small changes in the pattern of infection, fluctuating around 23% during these 6 years.

In spite of the low rate of infection with deep ploughing in the first period the level of take-all rose steeply from 21% to 49% in the 9th year to 61% and 86% in the 10th, when this treatment showed the greatest increase; there were no changes in the levels at the early sampling in the 10th and 11th years for the other treatments, but under deep ploughing infection decreased to their level in June of the 11th year.

Although the first increase in disease incidence was delayed under minimal cultivation, the main peak occurred at the same time and level as in the other treatments but decreased to a lower level in the 12th year.

Despite the decrease in incidence from August of the peak year to June in the 12th with all cultivations, a comparison between the June figures for both years shows a decrease only in the shallow ploughed and direct-drilled treatments. A comparison of August levels in the last 2 years shows a very substantial decrease with direct-drilling, a smaller decrease in shallow and chisel ploughed plots and none under the deep-ploughed treatment.

The results of adding 100 kg N/ha on the level of take-all under different cultivation treatments is shown in figures 8 and 9. The obvious effects were the smaller increases

Fig 8

The effect of cultivation treatments with 100 kg/ha of nitrogen fertiliser on the development of take-all at the early assessment

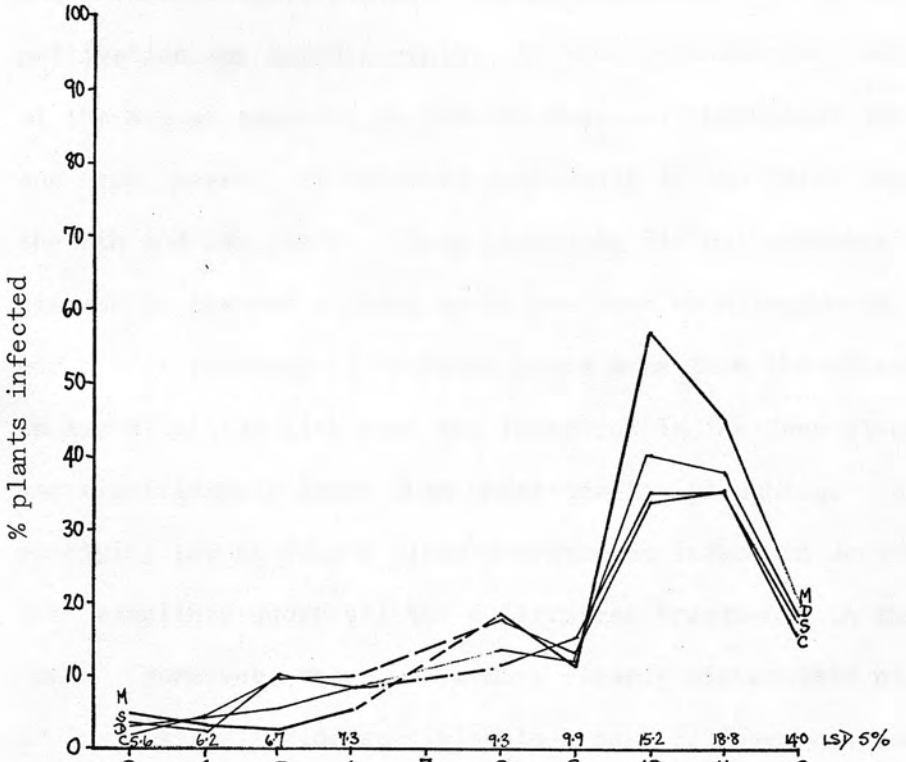
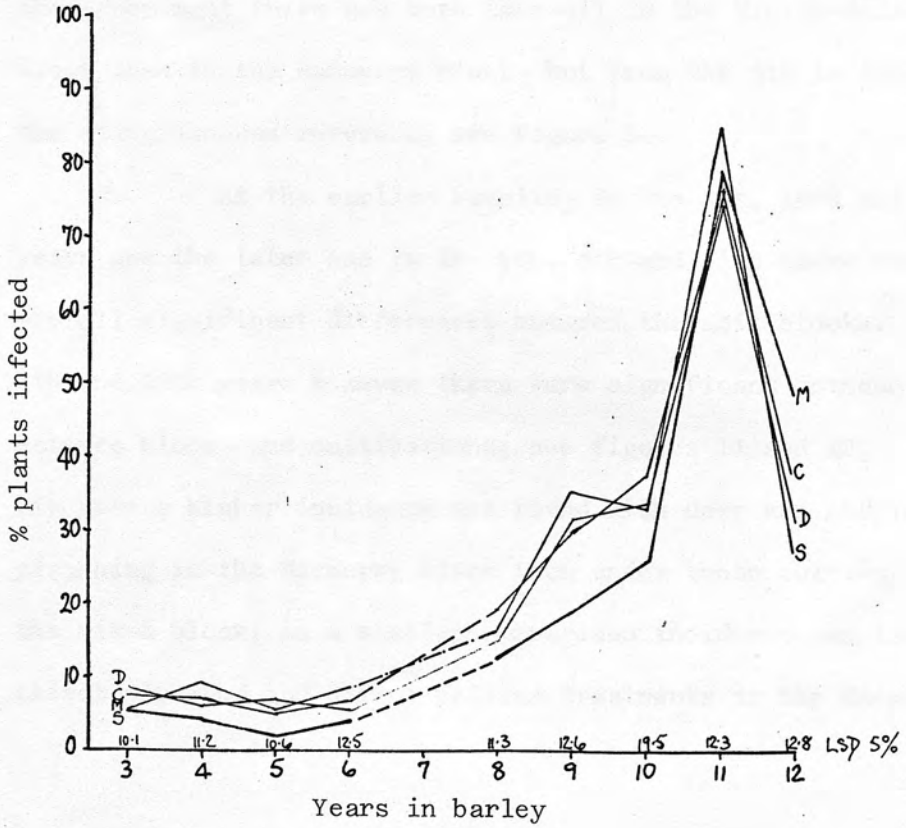


Fig 9

The effect of cultivation treatments with 100 kg/ha of nitrogen fertiliser on the development of take-all at the late assessment



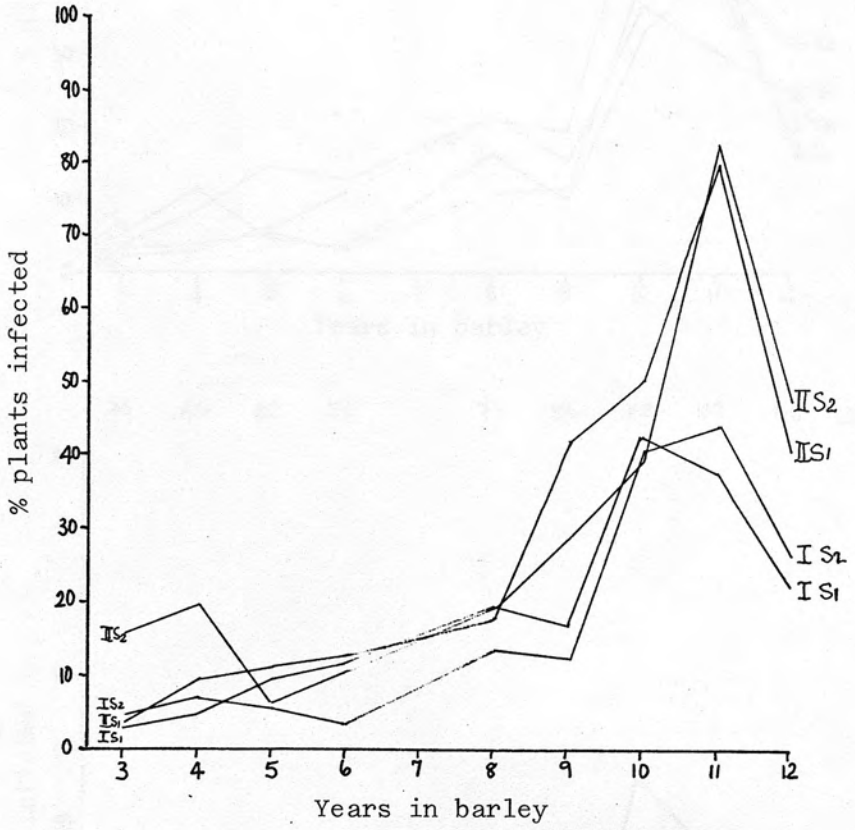
from the first to the second samplings in the early years and there was even a contraction in the 5th season in the shallow and chisel-ploughed plots. The suppressive effect of minimal cultivation was not discernible in the 3rd season but was seen at the August sampling in the 4th year and throughout the 5th and 6th year. It occurred again only in the later samples in the 8th and 9th years. Deep ploughing did not decrease take-all disease in the early years as it did when no nitrogen was applied, nor did it increase it in later years more than the others; indeed, in August of the 11th year the infection in the deep-ploughed plots was significantly lower than under shallow-ploughing. On plots receiving 100 kg N/ha a clear decrease in infection occurred at both samplings under all the cultivation treatments in the 12th year. Moreover, there was a more clearly discernible pattern of low disease incidence rising to a peak followed by a decline with this nitrogen treatment.

In the 3rd and 4th years and 11th and 12th years of the experiment there was more take-all in the Winton-Macmerry block than in the Macmerry block, but from the 5th to 10th years the situation was reversed; see figure 10.

At the earlier sampling in the 4th, 10th and 12th years and the later one in the 6th, 8th and 11th there were no overall significant differences between the soil blocks. In the 8th and 10th years however there were significant interactions between blocks and cultivations; see figures 11 and 12. In the 8th year a higher incidence was found with deep and shallow ploughing in the Macmerry block than under those cultivations in the mixed block; in a similar comparison incidence was lower under chisel ploughed and direct-drilled treatments in the Macmerry block.

Fig 10

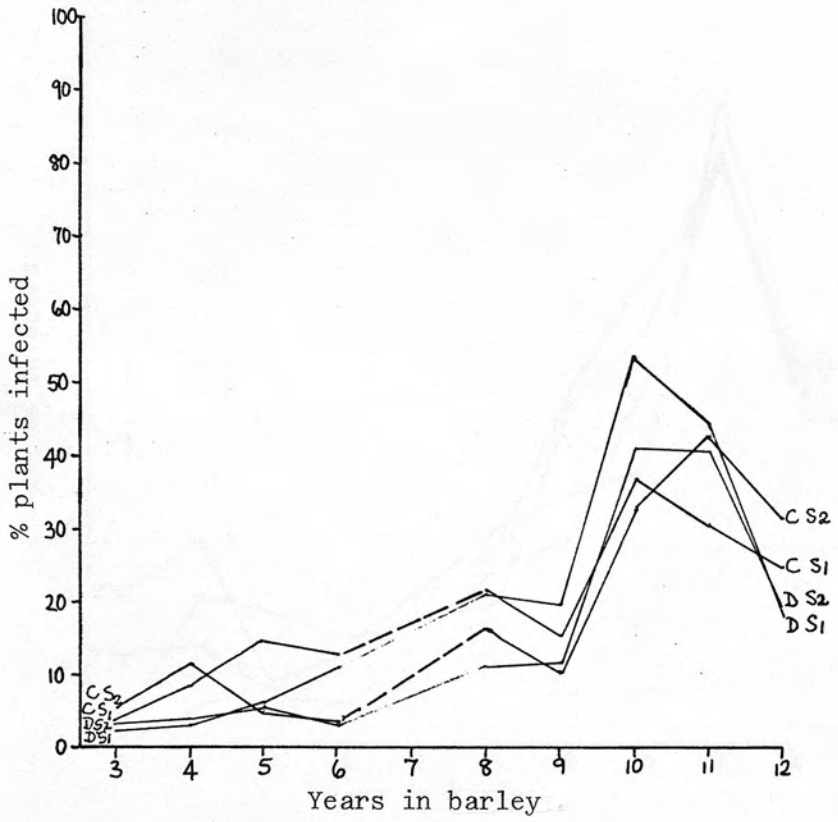
The effect of soil block on the development of take-all at early (I) and late (II) assessments.



I	1·2	2·1	2·0	2·6	4·1	4·1	5·3	5·1	5·5	LSD 5%
II	3·0	4·3	4·5	5·1	3·0	4·1	7·3	4·8	3·2	

Fig 11

The effect of soil block and cultivation on the development of take-all at the early assessment



2·5 4·6 4·2 5·4 7·5 8·6 11·2 10·9 11·6 LSD 5%

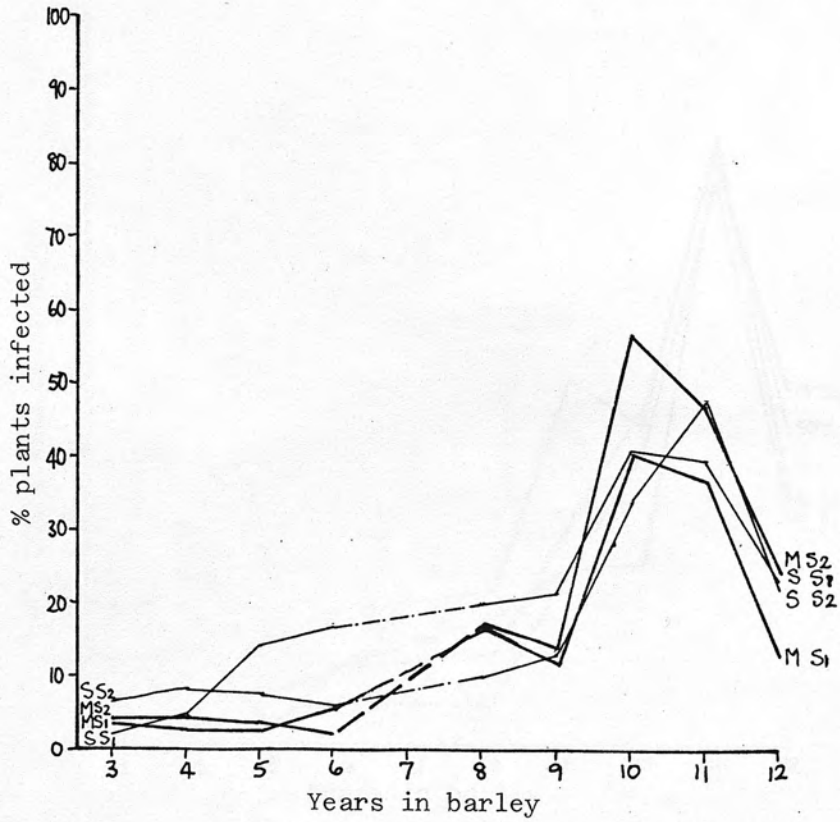
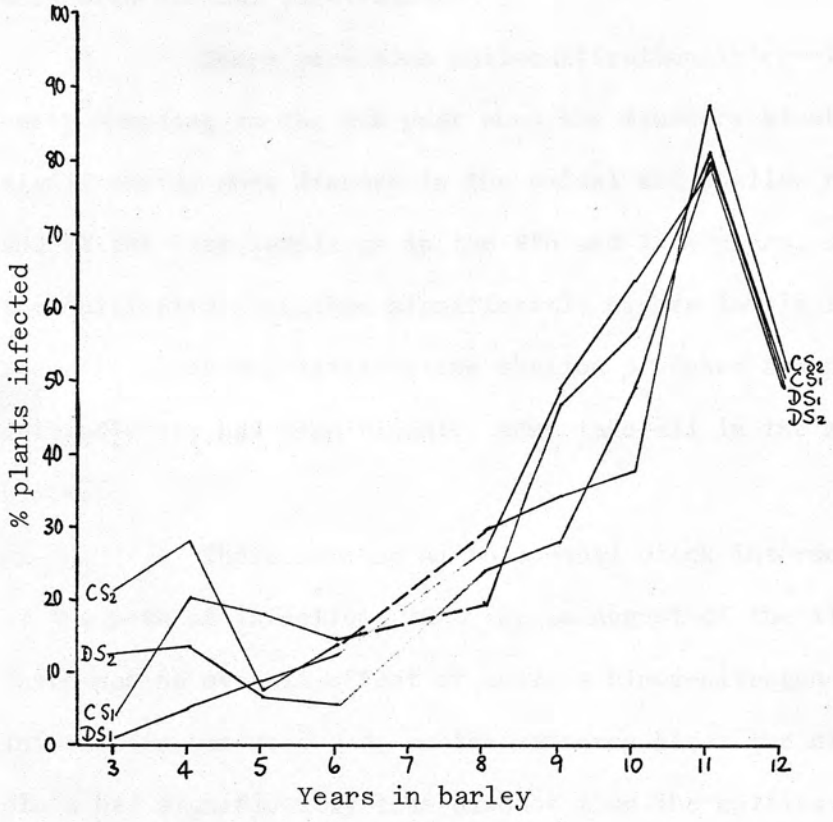
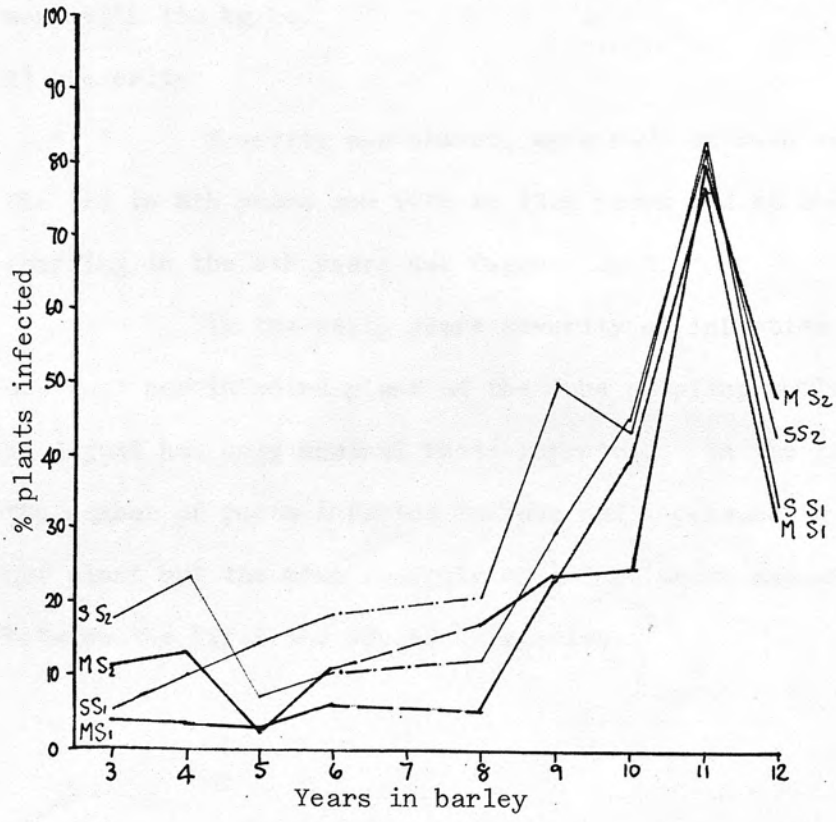


Fig 12

The effect of soil block and cultivation on the development of take-all at the late assessment



6·3 7·0 9·5 10·8 6·3 8·7 15·5 10·2 6·8 LSD 5%



At the early sampling in the 10th year a higher incidence was recorded only under deep-ploughing in that block and a lower only with minimal cultivation.

There were also soil-cultivation interactions at the early sampling in the 5th year when the Macmerry block had significantly more disease in the chisel and shallow ploughed plots and at the late samplings in the 9th and 12th years, when firstly the cultivated plots had significantly higher levels in the Macmerry block and latterly the shallow ploughed and direct-drilled plots had significantly more take-all in the mixed soil block.

There were no nitrogen-soil block interactions, but at the peak of infection, that is, in August of the 11th year when there was no overall effect of soil, a block-nitrogen-cultivation interaction was recorded: on the Macmerry block the direct-drilled plots had significantly less disease than the cultivated plots where no nitrogen or 50 kg/ha had been added and significantly more with 150 kg/ha.

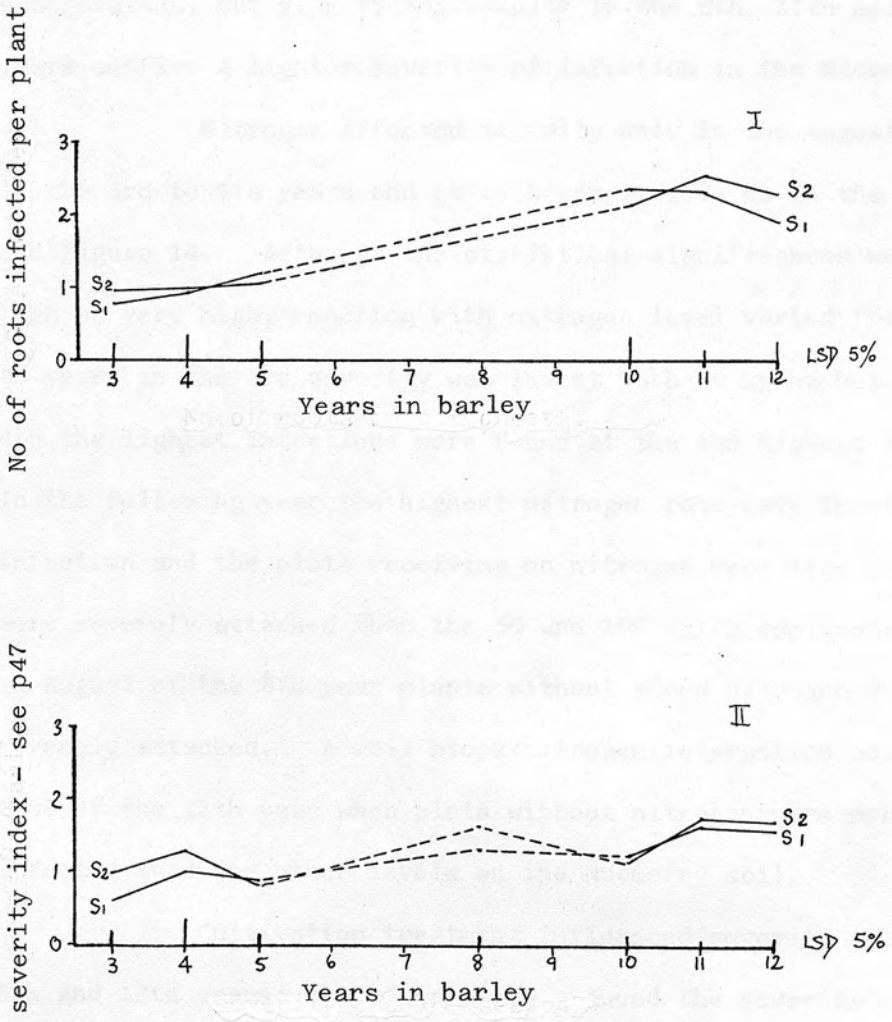
2) Severity

Severity assessments were made at both samplings in the 3rd to 5th years and 10th to 12th years and at the second sampling in the 8th year; see figure 13.

In the early years severity of infection was about one root per infected plant at the June sampling whilst most plants in August had only seminal roots infected. In the later years the number of roots infected in June had increased to more than two per plant but the mean severity of infection in August still fell between the first and second categories.

Fig 13

The effect of soil block on the severity of infection at the early (I) and late (II) assessments



Data from severity assessments could be analysed for soil, cultivation and nitrogen effects in the later years, when all plots were infected, but in the 3rd to 5th years samples from some plots were unaffected by take-all and statistical analyses for soil block differences were unreliable. The results for early years, when analysed for soil differences, included the uninfected plots; and are thus influenced by incidence. Bearing this in mind, the much higher severity index of the mixed Winton and Macmerry block compared with the Macmerry block in the 3rd year is probably an exaggeration, but significant results in the 8th, 11th and 12th years confirm a lighter severity of infection in the Macmerry soil.

Nitrogen affected severity only in the August samples in the 3rd to 5th years and at both sampling dates in the 10th year; see figure 14. Although the statistical significances were always high or very high, reaction with nitrogen level varied from year to year; in the 3rd severity was lowest with 50 kg/ha but in the 4th the lightest infections were found at the two highest levels. In the following year the highest nitrogen rate gave least severe infection and the plots receiving no nitrogen were significantly more severely attacked than the 50 and 100 kg/ha applications. In August of the 8th year plants without added nitrogen were most severely attacked. A soil block/nitrogen interaction occurred in June of the 12th year when plots without nitrogen were more heavily infected than the other levels on the Macmerry soil.

Cultivation treatment influenced severity only in the 5th and 12th years: direct-drilling reduced the severity compared with ploughing in June of the 5th, but by August it was only significantly less than the deep-cultivated plots; in the 12th year, however, all cultivated treatments were less severely infected than

Fig 14

The effect of nitrogen treatments on severity of infection at the early (I) and late (II) assessments

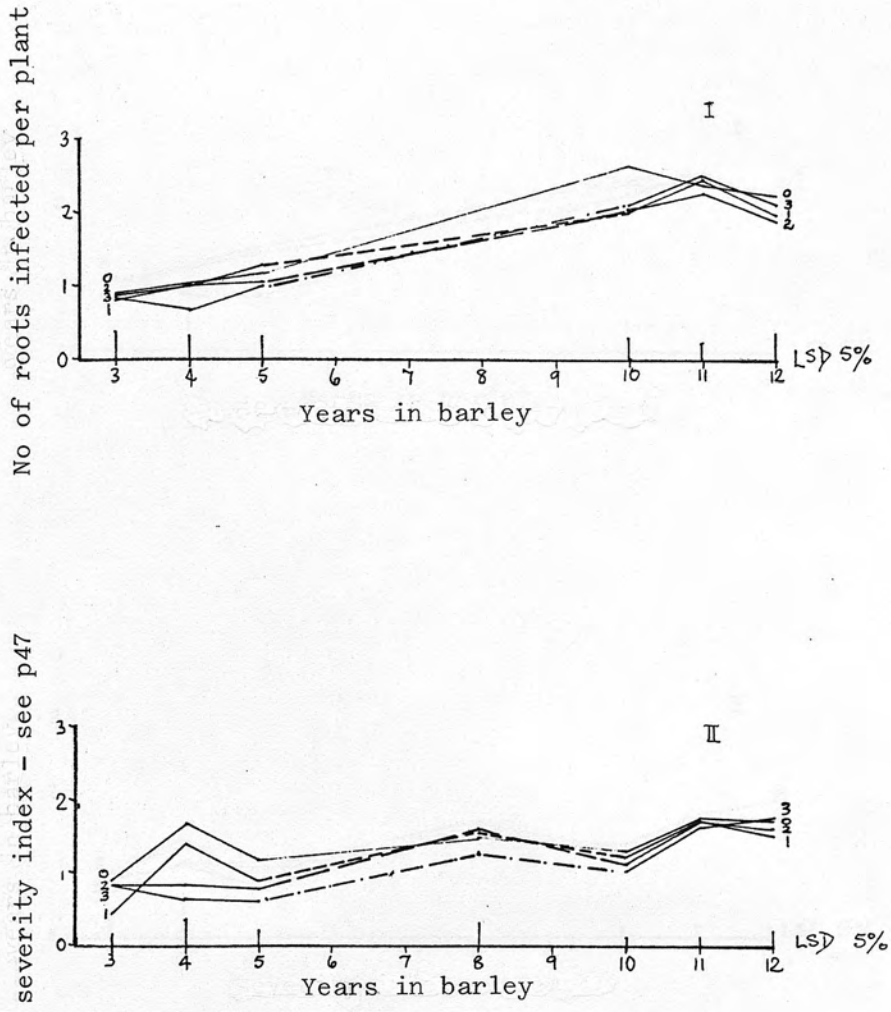
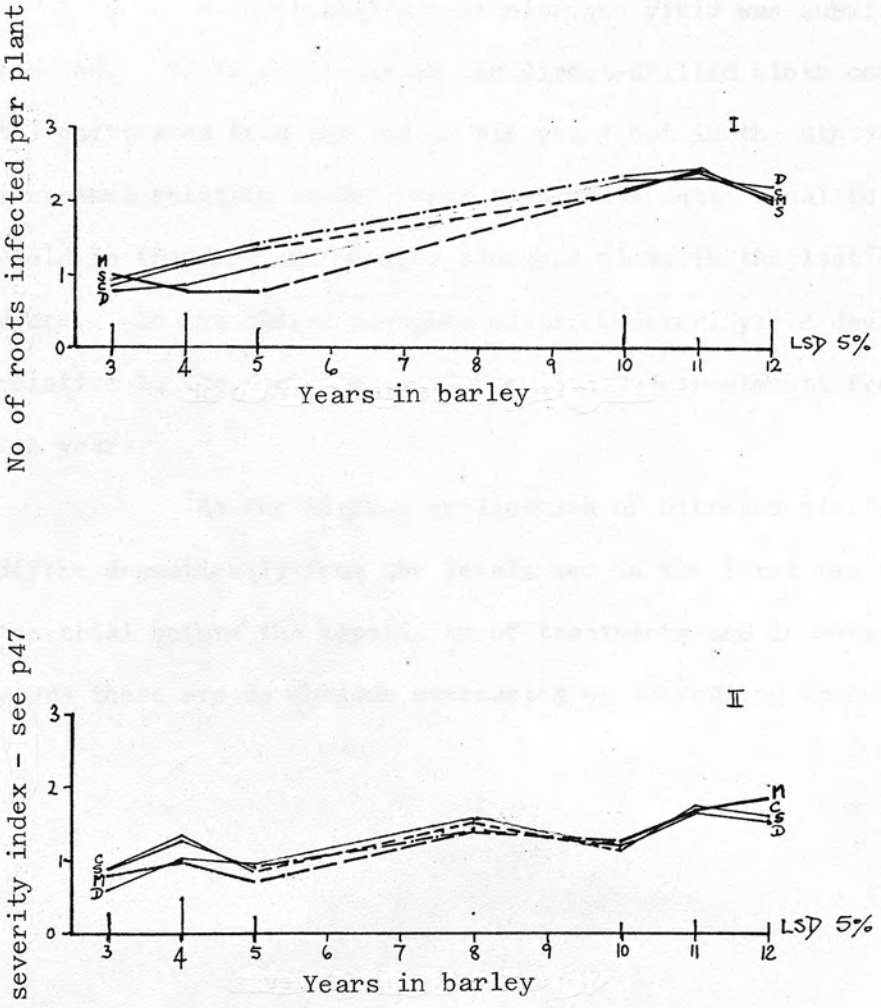


Fig 15

The effect of cultivation treatments on severity of infection at the early (I) and late (II) assessments



the unploughed in August. These results are shown in figure 15.

3) Yield

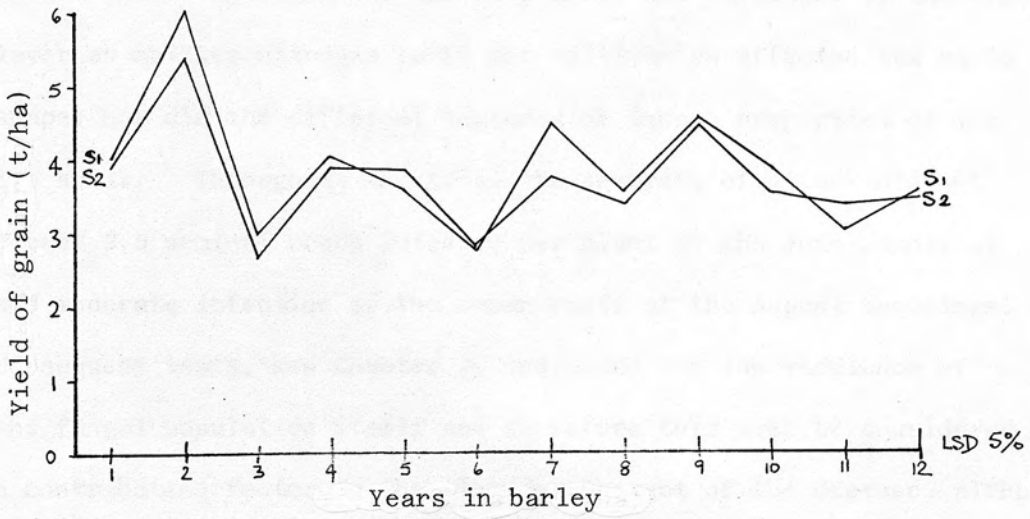
The annual yield of the South Road Experiment is shown in figure 16. In the first two years, before the imposition of treatments, the mean yields were 3.92 and 5.73 t/ha. The overall pattern fluctuated about the mean of the 3rd to 12th years: 3.55 t/ha. In the 1st, 2nd, 5th and 7th years there was a significant difference between the soil blocks, the Macmerry soil outyielding the mixed soil block, except in the 5th year.

Without addition of nitrogen yield was substantially reduced. Yield was lower in the direct-drilled plots compared with the cultivated from the 3rd to 8th years but in the 9th year it increased relative to the other treatments until equal to the yield in the deep and shallow ploughed plots in the last three years. In the chisel ploughed plots, however, yield declined relative to the deep and shallow cultivated treatments from the 7th year.

At the highest application of nitrogen yields did not differ dramatically from the levels set in the first two years of the trial before the imposition of treatments and in subsequent years there are no obvious decreasing or increasing trends.

Fig 16

The effect of soil block on yield of barley (t/ha) at 85% moisture from the South Road trial from 1966 to 1977



Discussion

General pattern of take-all development from the 3rd to 12th years.

The recording of a low incidence of take-all until the 9th cereal crop does not concur with the expected TAD pattern described by Shipton in 1967, although subsequently, in 1975, he reported a site where maximum disease occurred as late as the seventh consecutive crop because of an unaccountably slower development of take-all. The slow development in the South Road Trial might be attributed to one or more previously established biological, physical or chemical antagonistic factors in the soil, a generally low virulence of the population of G. graminis in the soil, or a combination of these. In addition, the fact that the host was barley, not wheat, may have influenced development.

The incidence pattern does not appear to be a function of the fertility status of the soil or of the structure of the upper layer as neither nitrogen level nor cultivation affected its basic shape; nor did the different textures or innate properties of the two soils. Throughout the trial the severity of attack did not exceed 2.5 seminal roots infected per plant at the June samplings and moderate infection of the crown roots at the August samplings. Subsequent tests, see Chapter 3, indicated the low virulence of the fungal population itself and therefore this must be considered a contributing factor to the slow development of the disease, although it did not ultimately prevent a high incidence.

The existence of an antagonistic factor such as found in Eastern Washington (Shipton, Cook and Sitton, 1973) but working only below a certain level of incidence, is possible except for the difficulty of defining the threshold: inspection of the results for

the 8th to 10th years under different cultivations without nitrogen reveals great variability in behaviour. Comparisons can be drawn between a theoretical basis for the existence of a threshold and the inoculum theory of Garrett (1970). In the same way that inoculum potential of the pathogen at any site must be adequate before infection becomes possible, so it might be argued here that a certain number of plants must be infected by late June before a widespread attack can develop later in the season, incidence being determined by a large area of host surface coming within the range of infection; or, to explain in Garrett's words, adequate incidence being equivalent to "adequate cross-sectional area of the fungus in contact with a unit area of host surface". The low virulence of the pathogenic population reflects the degree of vigour of the infecting hyphae as described by Garrett except that virulence was not affected by nutrient status of the fungal population to the extent of affecting the pattern of infection. The third factor affecting inoculum potential is the collective effect of environmental conditions, whose effect on this trial is discussed in the later part of this section.

In the earlier years the possibility of biological control by Phialophora radicicola Cain var graminicola Deacon, as first suggested by Scott (1970) and Balis (1970) and subsequently investigated by Deacon (1973 a and b; 1976), was discounted by the latter (pers comm, 1977) because the cropping history of the field had not been suitable for the establishment of a population of this fungus. The cropping history is given in Appendix I.

In the South Road trial the peak of take-all incidence in the 11th year coincided with the low rainfall and high 5 cm soil temperatures in June and July; there were also low rainfall and

relatively high temperatures when accumulated from the preceding December to February but these latter conditions also occurred during the winter before the 8th season when infection was low. The increases in infection between the late June and early August samplings were not apparently correlated with rainfall or temperature of July, nor June plus July, but it is possible to see some relationship between the ultimate level of take-all each year and climatic pattern of these two months in that peaks of incidence occurred when high soil temperature coincided with low rainfall; see figure 17.

This response is not in keeping with the literature and experience in cereal growing areas of the USA (Cook, Papendick and Griffen, 1972); so the climate may have had a more important influence on some factor other than the fungus itself. It is possible that dry conditions depressed the general microbial level of the soil and allowed take-all to spread as it could not otherwise, thus equating the reaction with temperature to Henry's (1932) and Garrett's (1934) findings in sterile soil. If this is correct it also provides incidental evidence of the low competitive character of the population of *C. graminis* in this trial.

Factors affecting take-all incidence from the 3rd to 6th years.

The reduction of take-all incidence in uncultivated plots when compared with shallow and chisel-ploughed areas was expected (Hood, 1965; Brooks and Dawson, 1968). Soane (1969) measured soil bulk density, and therefore porosity, under the different cultivations in 1968, and found high bulk density in the top horizon of the uncultivated plots; see figure 18. Organic matter accumulates in direct-drilled plots resulting in a high

Fig 17

The development of take-all at the late assessment and the accumulated rainfall and 5 cm soil temperatures of June and July from 1968 to 1977

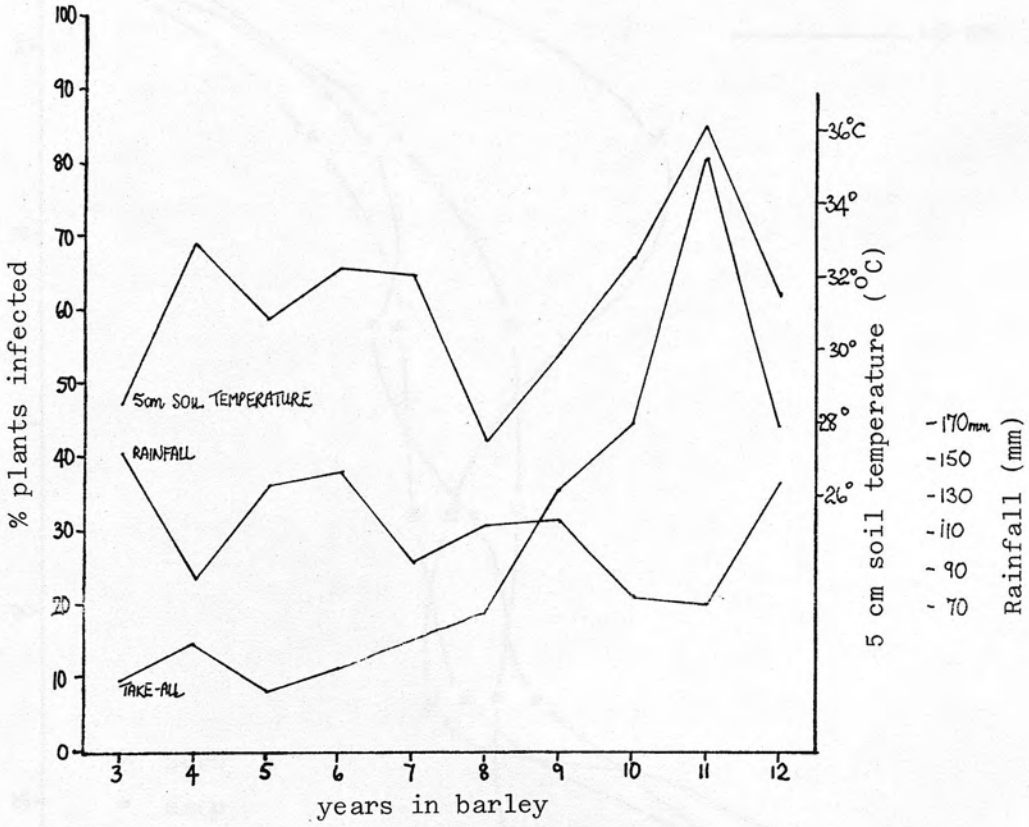
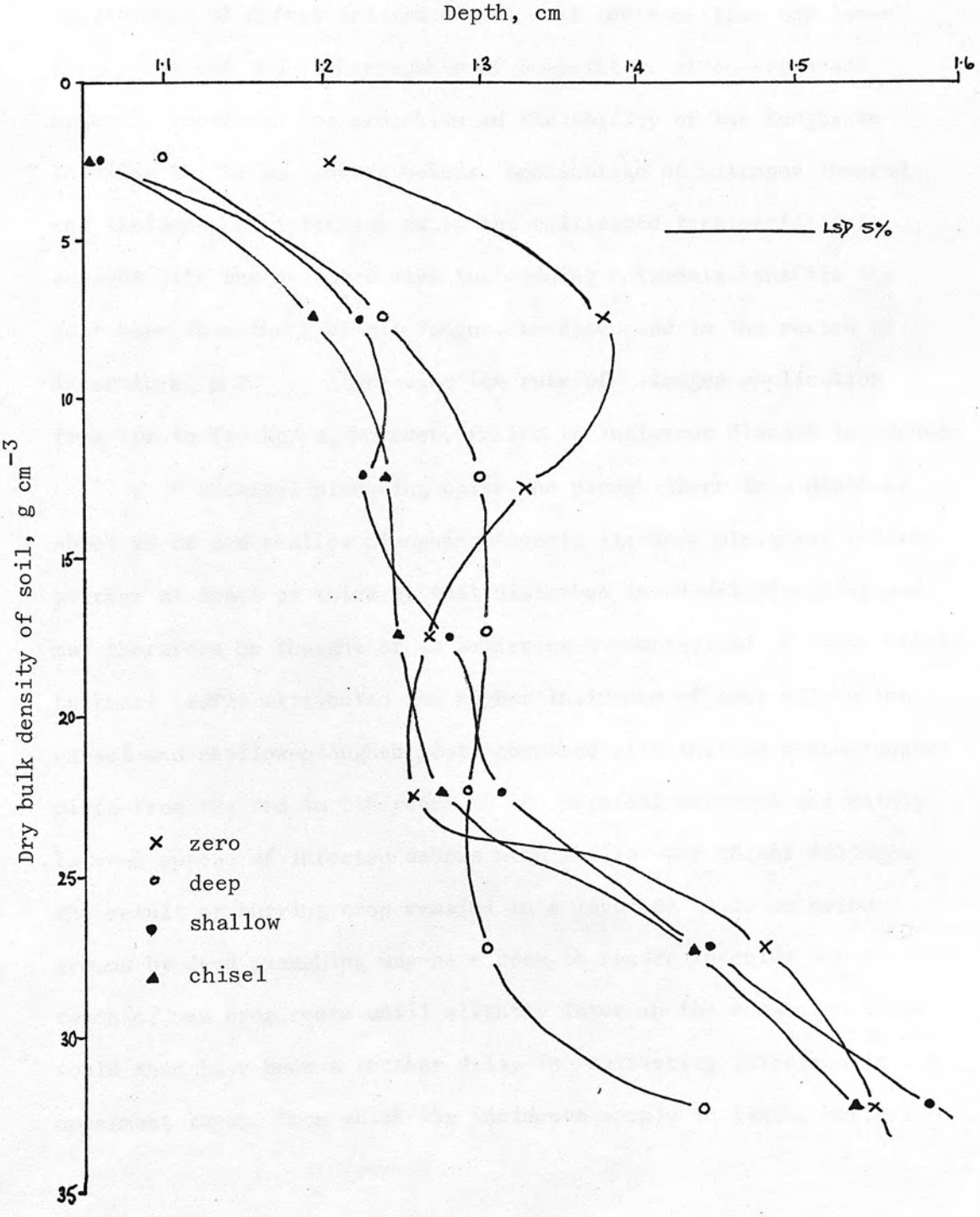


Fig 18

The variation of dry bulk density with depth at the time of emergence of barley for deep, shallow, chisel and zero ploughing in South Road Field in 1968. (Soane, 1969)



micro-organism population (Nishita, Kowalewsky and Larson, 1956) and mineralisation of organic nitrogen can be slower (Arnott and Clement, 1966; Dowdell and Cannell, 1975). It is therefore probable that competitive saprophytic organisms make applied nitrogen less available to the fungus and the host plants for much of the season and this may account for the higher nitrogen requirement of direct-drilled crops. At the same time the lower oxygen content and higher number of competitive micro-organisms probably result in the reduction in the ability of the fungus to colonise the host. Nevertheless, application of nitrogen lowered the incidence of infection as in the cultivated treatments; this accords with the accepted view that adding nutrients benefits the host more than the take-all fungus, as discussed in the review of literature, p 20. Increasing the rate of nitrogen application from 100 to 150 kg/ha, however, failed to influence disease incidence.

Chisel ploughing opens the plough layer to a depth of about 25 cm and shallow ploughing inverts it; deep ploughing inverts a layer at least as thick as that disturbed in chisel ploughing and may therefore be thought of as achieving a combination of these effects. Lockhart (1972) attributed the higher incidence of take-all in the chisel and shallow-ploughed plots compared with that in deep-ploughed plots from the 3rd to 5th years to the physical movement and mainly lateral spread of infected debris with shallow and chisel tillage. The result of burying crop remains in a layer 25 to 30 cm below ground by deep ploughing may have been to render inoculum out of reach of new crop roots until slightly later in the season. There would then have been a further delay in registering infection in the uppermost layer, from which the incidence sample is taken, because

the infecting fungi would have farther to grow. Because of a low initial infection in the earliest years it may be that a large proportion of the debris was subject to saprophytic break-down for 16 months instead of 4 months. Furthermore, the greater soil aeration at a depth of 27.5 and 32.5 cm in deep-ploughed plots compared with other treatments may have resulted in a more rapid breakdown of debris. It is possible that inspection of roots throughout the plough layer would have revealed more infections.

There was an increase in incidence in the 4th year under chisel and shallow ploughing in the August samples at the mean of nitrogen levels and at both samplings when no nitrogen was applied. A similar increase occurred only at the late sampling without nitrogen in the deep-ploughed plots, whereas the level in the direct-drilled plots was completely unaffected that year. This suggests that conditions responsible for the increase in the cultivated plots were absent or their effects suppressed in the undisturbed plots. The deep-ploughed plots showed an increase only when no nitrogen was added and the rate of breakdown of debris diminished.

When 100 and 150 kg/ha of nitrogen were applied no differences occurred in disease level between the cultivations except in the 5th year when the uncultivated plots at the early sampling had significantly less take-all than the chisel and shallow ploughed. These higher levels of nitrogen completely suppressed the 4th year increase discussed above, thus indicating the importance of nutrition in the host-soil microcosm relationship.

The effect of soil type, however, appeared to be independent of nitrogen treatment but often interacted with cultivation. However, there was an alternation of higher incidence of disease level

between the mixed and Macmerry blocks and complicated changes of relationship in interaction between soil type and cultivation treatment over the years with an absence of any constant pattern or trend. Variation in weather conditions from year to year may have accounted for this.

Factors affecting take-all incidence from the 8th to 12th years.

By the end of the 8th season there was evidence of the beginning of a substantial increase in take-all incidence on many plots: without added nitrogen the disease in the deep- and chisel-ploughed plots with their greater depth of soil disturbance than the shallow-cultivated plots, and therefore greater aeration, had markedly increased. Lack of applied nitrogen would result in fewer micro-organisms and although there would have been less organic matter input because of decreased plant and root numbers, breakdown of residues would be less. Another result of lack of applied nitrogen was the presence of more couch grass, the rhizomes of which provide a parasitic bridge between crops (Zogg, 1963). Despite the fact that the greater aeration in the deep- and chisel-ploughed treatments would cause more oxidation of organic matter the balance between all factors was in favour of fungal colonisation.

Although the level of incidence of disease in the shallow-ploughed plots rose to equal that of the other cultivated treatments in the 9th season, the suppressive effect of direct-drilling retarded the increase in this treatment to the 11th season.

It was not until the 10th year that an increase was recorded in the early sample, where, without addition of nitrogen, incidence rose to about 30% in all cultivation treatments. At that point an unexpected interaction of nitrogen with deep and minimal cultivation occurred: without nitrogen the level of disease in the

deep-ploughed plots was greater than in the other cultivated plots and much greater than in the uncultivated ones; yet with 100 and 150 kg/ha of nitrogen the direct-drilled plots had significantly more disease than the cultivated. It can only be surmised that since adding nitrogen did not reduce survival of disease as found until the 8th season, in this instance the addition of the higher levels of nitrogen allowed the fungus to overcome the suppressive effect of direct drilling after overwintering. A possible mechanism for this effect could be that if the micro-organism population was maximum in direct-drilled plots there would be less competition for added nitrogen and the already rapidly increasing population of G. graminis could take full advantage of it.

SEVERITY

The low disease severity throughout the trial was noted in the opening discussion of this experiment. Cunningham (1975) recorded a relationship between incidence and severity, but here the small changes in severity that occurred did not follow the incidence pattern.

The use of barley rather than wheat as a host crop may partly explain the low severity rating, barley showing a greater ability to regenerate new root growth and offset the effects of infection (Cunningham, 1967b; Asher, 1972a).

The greater severity of infection on the plants in the mixed soil block compared with the Macmerry block cannot easily be ascribed to one soil factor as severity depends on host susceptibility and rate of growth of parasite up the root, both internally and externally. Host susceptibility to take-all is influenced by level of nitrogen nutrition but rate of external growth is affected by

competition and overall environmental conditions of the rhizosphere as well as virulence of infecting strain. Virulence was not found to differ with populations of strains from the different soils; see chapter 3. Environmental conditions in the Macmerry block would theoretically be more suitable for growth of the take-all fungus than in the mixed soil block because of its higher sand content (Garrett, 1934). Susceptibility of host did not appear to be related to soil type as there were no interactions between soil blocks at any nitrogen level at any sampling. Severity, however, was affected by nitrogen level: the level at which lightest infections occurred varied with year and therefore, presumably, with complex climatic factors, but was never in the plots which received no nitrogen. Huber, Painter, McKay and Peterson (1968) reported increased activity of G. graminis, but reduced disease severity, with increasing levels of nitrogen as ammonium sulphate and ammonium nitrate; the reduction presumably resulting from an increase in competition in the rhizosphere since the use of both forms of nitrogen radical precludes discussion of the possible effect of complicated ammonium:nitrate ratios.

Weste and Thrower (1971), working in vitro with Garrett's Cambridge method of wheat inoculation (1970) found that the addition of nitrate increased pathogenicity of virulent isolates more than it stimulated root production by the host. From this it can be inferred that if contemporary levels of applied nitrogen could by-pass the competitive organisms in the rhizosphere the commonly found reaction of take-all with nitrogen addition would be reversed. This idea is supported by Morrison's findings (1976) that in sterilised soil highest yields of cereals were achieved with approximately half the level of nitrogen required in unsterilised soil.

YIELD

From figure 16 there is little evidence of yield loss associated with take-all except in the 10th, 11th and perhaps 12th crop. This means that there was a lack of effect of disease on yield until over 50% of plants had been infected.

It could be argued that take-all never influenced yield, even at the peak of disease in 1976, the 11th year; yet it seems unlikely that in a year of great drought the loss of roots in the upper soil layer of 80% of the plants could have failed to have some effect on yield. In water culture Humphries (1958) found that removal of up to 5% of roots of barley and rye had no effect on the growth of the root system, but that rate of shoot growth decreased with increasing amount of root removal; he conjectured that removal of over 50% of roots decreased mineral supply to the root system and root growth rate. On average the plants were not affected to this degree.

Because of the obvious lack of yield effect in most years the data were not subjected to a regression analysis in the manner of Scott and Hollins' work with eyespot (1978).

Shipton (1975) recorded a site where take-all had negligible effect on barley yield; indeed it is common to find a much lower yield loss with take-all in barley crops than in wheat (Cunningham, 1967b; Cunningham, Spillane, Foreman and Conniffe, 1968; Jensen and Jorgensen, 1973; Shipton, 1975; Jepsen and Jensen, 1976). Asher (1972a) found that barley responded to infection by producing more new roots than wheat and suggested that this accounted for the apparent difference in disease tolerance. From this it can be surmised that if winter or spring wheat had been grown continuously on this site a more obvious relationship of yield with take-all incidence may have been seen.

CHAPTER 2

TAKE-ALL DEVELOPMENT EXPERIMENTS

A series of glasshouse experiments was designed to study the possible changes in levels of take-all infection with time using different soils. It was initiated because of the unexpected pattern of disease development found in the South Road trial.

Infection patterns were followed in four experiments:

1 continuous cropping of soil from the South Road trial area;

2 continuous cropping of four contrasting soils with different histories and subjected to various treatments;

3 a similar experiment to the second except using three soils and comparing the effects of disturbing and not disturbing the soil;

4 continuous cropping of miscellaneous soils.

Basic materials and methods

Soil was collected, potted as soon as possible and planted with wheat or barley seed which had not been treated with fungicide. After 7 days' growth each pot was given 10 ml of a nutrient solution containing 0.2 g nitrogen, and 0.025 g each of phosphorus and potassium per litre.

After 4 weeks' growth pots were removed for the measurement of number of roots and length of infected roots of the plants. Plants in the remaining pots were cut at seed level and the pots were resown; removal, replanting and feeding continued every 28 days. The pots were stood in plastic seed trays and placed in a sunken gravel bed which had facilities for flood-watering and supplementary light by sodium lamps at a height of approximately 1 m to produce an 18-hour day when necessary; pots were also watered manually from above.

Temperature in the glasshouse was about 18°C in winter and spring but there was no means of ventilation apart from automatic roof vents and temperatures often rose above 30°C in summer and early autumn.

EXPERIMENT 1: TAKE-ALL INCIDENCE IN SOUTH ROAD TRIAL SOIL CONTINUOUSLY CROPPED EVERY 4 WEEKS WITH SEEDLING BARLEY.

Method: Soil from the Macmerry block of the South Road trial was removed on 28th March, 1975 before the trial for that year was planted and the take-all fungus could begin parasitic growth. Samples were dug from five pre-determined random places from the plots given annual dressings of 0 and 100 kg nitrogen per hectare on all four cultivation treatments; there were four replicates. The samples were potted in 250 ml plastic pots as described above and sown with six seeds of spring barley, cv Zephyr. At each harvest four pots per treatment were removed for assessment. The thirty-two pots of each harvest group were labelled with the number of their original field plot and laid out according to the South Road trial design; see Appendix I. Each group was then assigned a harvest number at random. After every replanting the remaining groups were relocated according to a new randomisation.

Results: Results only exist for harvests 4 to 10 as many of the pots from the first three harvests were lost as a result of excessive heat and the predation of mice and birds; only 5 pots were lost from subsequent harvests.

Throughout the experiment the level of infection in soil from the direct-drilled plots was very significantly lower than that in the cultivated soil and overall infection was higher in soil which had not habitually had applications of nitrogen in the trial.

Further analysis revealed that this was caused by a much smaller development of infection in the direct-drilled soil which had had nitrogen compared with that which had not; that is, that there was no difference in overall infection between soils which had not had nitrogen in the field irrespective of cultivation treatment; see table 1.

TABLE 1 : MEAN TAKE-ALL INFECTION (cm/root) OVER ALL HARVESTS

Cultivation	Nitrogen		Mean
	-	+	
Deep	3.75	2.95	3.35
Chisel	3.21	3.97	3.59
Shallow	3.94	3.26	3.60
Minimal	3.11	0.66	1.88
Mean	3.50	2.71	3.11

SE (difference)	nitrogen level	± 0.327
	cultivations	± 0.314
cultivations v nitrogen level:	cultivations	± 0.654
	nitrogen level	± 0.559

The general harvest pattern showed a significant decrease in infection at the 10th harvest compared with the 4th to 8th; see figure 19. Nitrogen treatment but not cultivation interacted with time; see figures 20 and 21. Soil from the plots without nitrogen gave a higher rate of infection until the 8th harvest when levels of the two nitrogen treatments were similar and both tended to decline. There was no interaction of nitrogen and cultivation with harvest.

Discussion: It can be seen from the pattern of development of take-all infection in the South Road trial from its 3rd to 12th year that when soil was collected for this experiment the level of incidence was still rising and there was evidence of suppression of the disease in minimally cultivated plots. In this

Fig 19

The development of take-all, mean of previous nitrogen and cultivation treatment

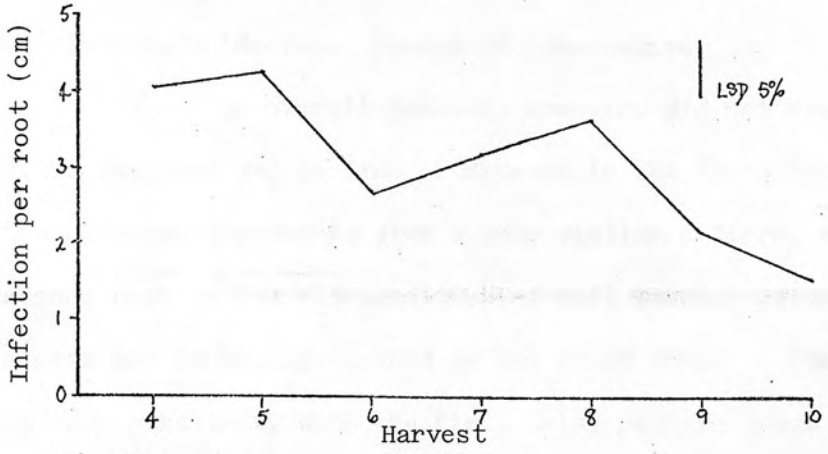


Fig 20

The effect of previous nitrogen treatments on development of take-all

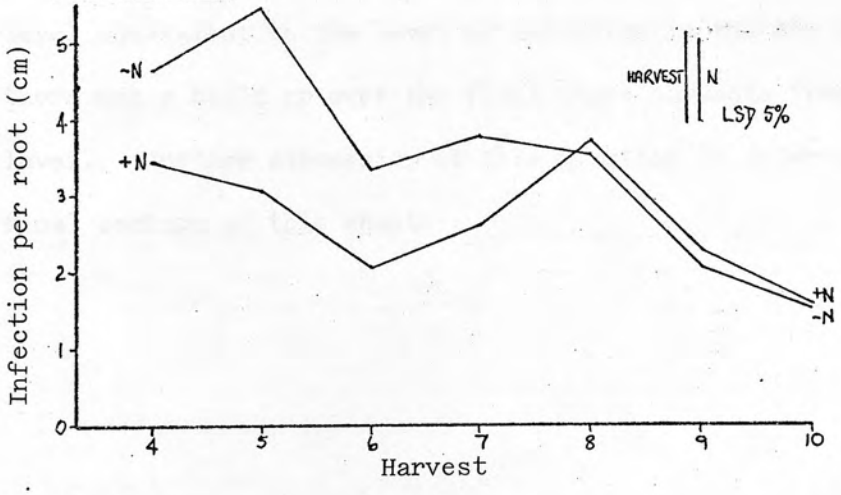
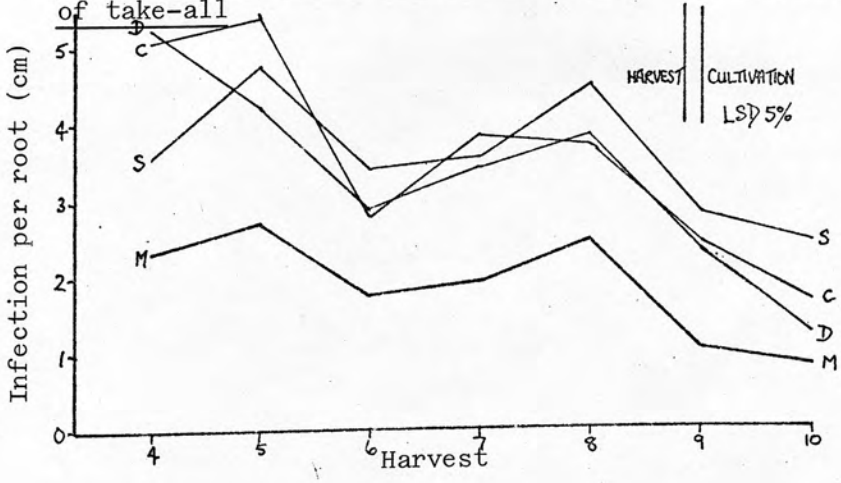


Fig 21

The effect of previous cultivation treatments on development of take-all



experiment the results showed that minimal cultivation with addition of nitrogen decreased the expression of infectivity; as this reflected the field pattern the decrease of infection in all treatments which occurred in the last two harvests can be tentatively ascribed to a 'decline' factor of some nature.

The overall pattern, however, did not straightforwardly mimic the peak and decline of disease in the field trial, nor did the nitrogen treatments give a very similar pattern, although the second peak in the nitrogen-treated soil samples could represent a peak yet to be experienced in the field trial. The lack of obvious similarity with the field trial pattern leads to the question of the relationship between them: without the first three harvests it is impossible to tell whether the in vitro pattern started at a level equivalent to the level of infection in the 8th or whether there was a build up over the first three harvests from a very low level. Further discussion of this question is deferred until the final section of this chapter.

EXPERIMENT 2: A COMPARISON OF INFECTION PATTERNS FOUND IN FOUR SOIL TYPES, EACH WITH FOUR CROPPING HISTORIES AND SUBJECTED TO FOUR TREATMENTS

This experiment investigated the possibility that different soils, when subjected to certain treatments might give distinctive take-all infection patterns according to their physical characteristics and cropping history.

Materials and methods

The soil series chosen for this experiment were:

Peffer (PF) - a sand

Hobkirk (HK) - a sandy loam

Kilmarnock (KK) - a clay loam

Whitsome (WH) - a clay.

Further details of these soils are given in Appendix II.

Soils were collected from fields with the following cropping histories:

H1 - no immediately previous cereal crop

H2 - 1 previous cereal crop

H3 - 3 previous cereal crops

H4 - 4 previous cereal crops.

The four treatments were:

1 - no addition of inoculum,

2 - addition of inoculum of G. graminis after first harvest,

3 - cropping with swedes until no infection occurs, then inoculated as 2,

4 - partial heat sterilisation, then as treatment 2.

The first treatment was a control which assessed natural inoculum; the second treatment sought to compare the reactions of the soils and histories to the addition of a known amount of parasitic

inoculum. The third and fourth treatments involved attempts to either exhaust natural inoculum in soil or partially sterilise the soil. By cropping with swedes it was hoped to determine how long different soils and histories took to lose the infectiveness of the indigenous G. graminis and to then assess the subsequent behaviour of added inoculum. By partial heat sterilisation it was hoped to kill non-spore-forming bacteria, thought by Shipton et al (1973) to be possible candidates for the TAD factor, and thereafter follow the behaviour of inoculum as before.

Inoculum was cultured from a piece of barley root infected by G. graminis grown in soil from the South Road trial. The root was surface sterilised for 5 minutes in a 10% solution of Deosan (hypochlorite) and then plated on to potato dextrose agar (PDA) with streptomycin. After about 7 days 0.8 cm^2 pieces of colonised agar were transferred to new PDA plus streptomycin plates. When the new colony had achieved a diameter of approximately 6 cm it was placed about 1.5 to 2 cm from the bottom of a pot of heat sterilised sand; six wheat seeds, cv Mega, were planted in the pot and after 28 days' growth the resulting infected wheat roots were cut into 1 cm lengths. For each experimental pot five pieces of root thus colonised were used.

To achieve partial sterilisation the soil sample was spread out to a thin layer in a metal tray and placed for 30 minutes in an oven preheated to $60^{o}C$.

The pots were cropped 8 times, in the manner described in the basic method, with 2 pots per treatment combination. The experiment was designed as four incomplete blocks. The main plots were harvests and within each the four soils were randomised with two of the histories and two of the experimental treatments.

The variability of the data increased with increasing length of infected root per root and in order to perform a valid analysis of variance the raw data, length of infected root per root, was transformed by $\sqrt{\text{raw data} + \frac{1}{2}}$; it is in this form that it is presented here.

Results

The pattern of infection per root over all soils, histories and treatments is illustrated in figure 22; it shows a gradual increase in infection to a maximum in the 4th harvest followed by a decline to a constant level in the last three harvests.

As the swede treatment was so different in nature from the other three it will be referred to separately at the end of this section. Overall infection was less in the control treatment than in the inoculated and partially sterilised/inoculated treatments; between the latter there was no significant difference. The Kilmarnock clay loam soil had significantly less take-all than the other three soils and the samples from fields after 3 years of cereals showed the greatest infection. Tables of results are given in Appendix II.

The treatments behaved similarly in each soil; the levels of infection found in the different soils for each history over all harvests is given in Table 2.

Fig 22

The development of take-all, mean of soils, histories and treatments

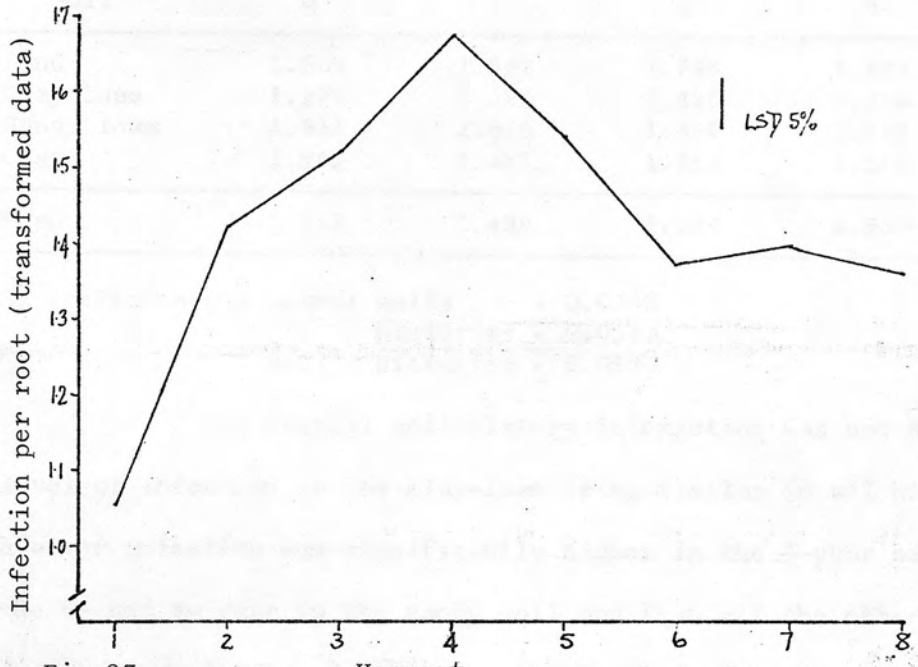


Fig 23

The development of take-all, in treatments 1, 2 and 4

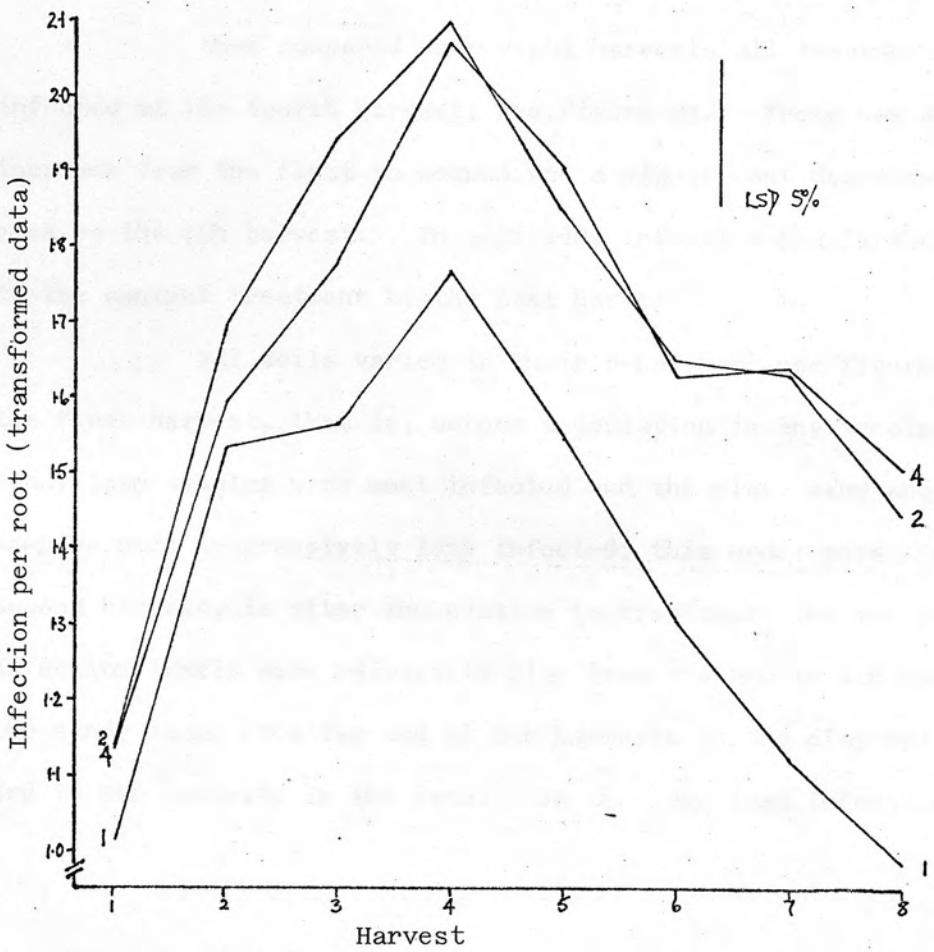


TABLE 2: TAKE-ALL INFECTION, MEAN OF HARVESTS AND TREATMENTS 1, 2 AND 4 (transformed data)

Soil	History (years in cereals) 0	1	3	4	Mean
Sand	1.556	1.692	1.794	1.523	1.641
Clay loam	1.277	1.428	1.420	1.318	1.361
Sandy loam	1.551	1.415	1.920	1.612	1.625
Clay	1.659	1.421	1.763	1.679	1.631
Mean	1.511	1.489	1.724	1.533	1.564

SE (difference): means: soils $\pm$ 0.0340
histories $\pm$ 0.0340
soil v histories $\pm$ 0.0680

The overall soil-history interaction was not strong, the level of infection in the clay-loam being similar in all histories; however infection was significantly higher in the 3-year history than the 0- and 4- year in the sandy soil and than all the other histories in the sandy loam. In the clay the level in the 1- year samples was significantly lower than in the other histories.

When compared over eight harvests all treatments were most infected at the fourth harvest; see figure 23. There was a significant increase from the first to second and a significant decrease from the peak by the 6th harvest. In addition, infection had further decreased in the control treatment by the last harvest.

All soils varied in their behaviour; see figure 24. At the first harvest, that is, before inoculation in any treatment, the sandy-loam samples were most infected and the clay, sand and clay loam samples were progressively less infected; this order persisted to the second harvest, ie after inoculation in treatments two and four. Infection levels were relatively high from the 2nd to 4th harvests in the sandy loam, from the 2nd to 5th harvests in the clay and from the 3rd to 5th harvests in the sand. In the clay loam infection rose

Fig 24

The effect of soil on development of take-all

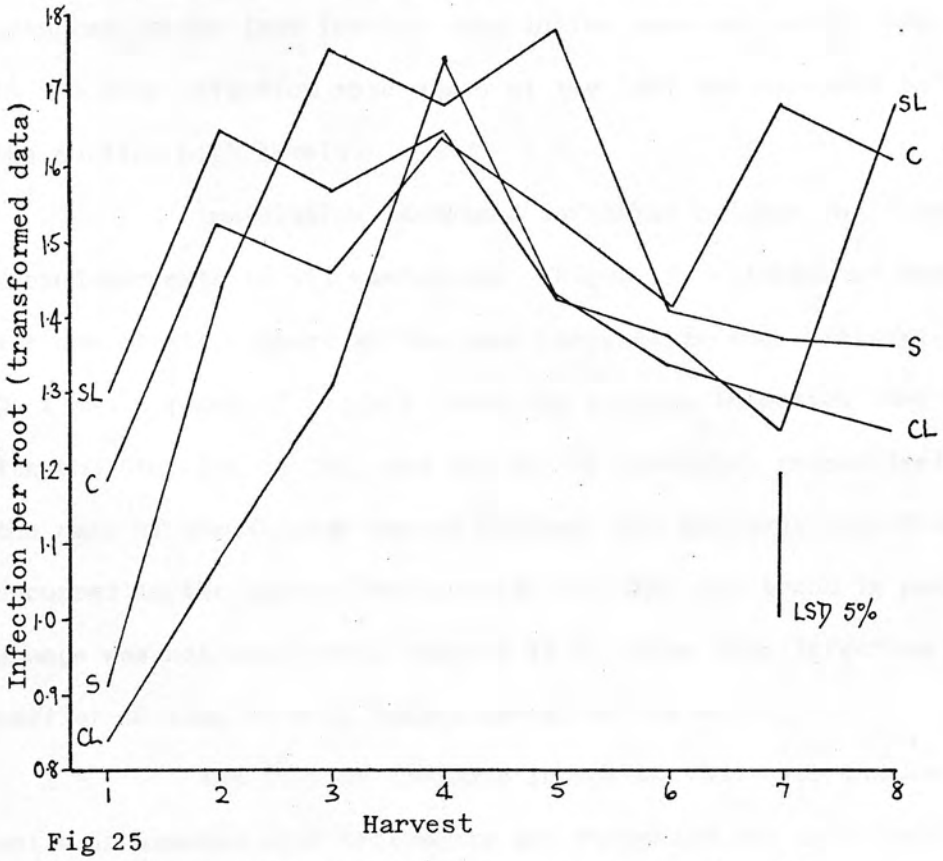
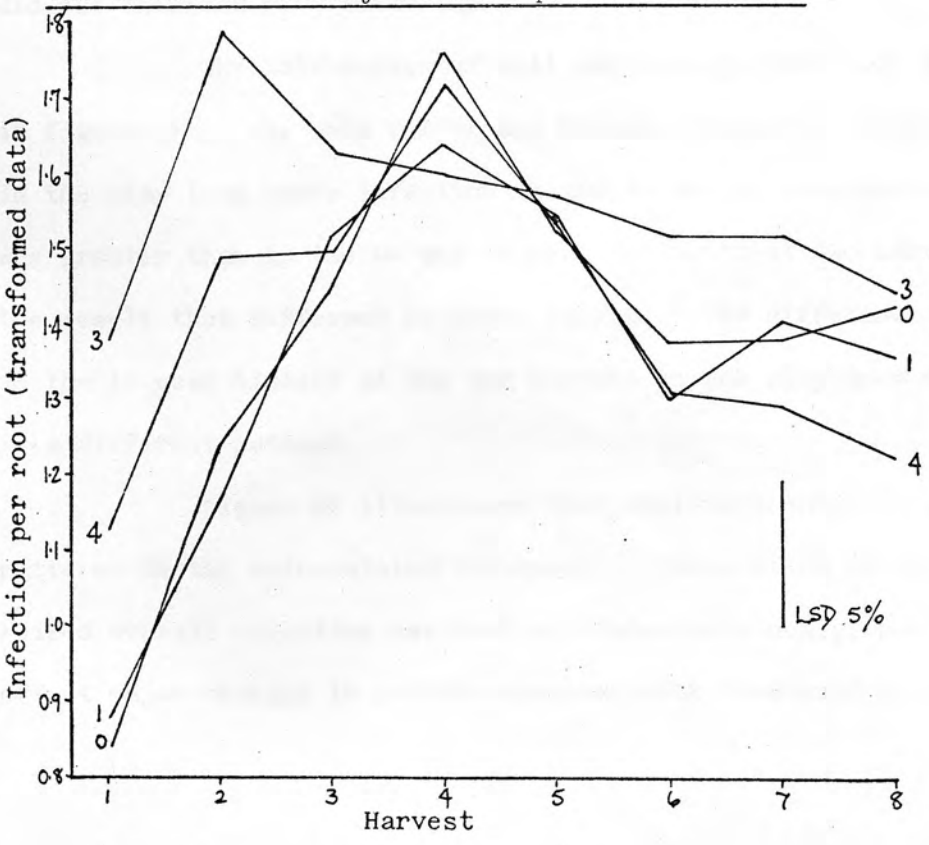


Fig 25

The effect of history on development of take-all



steadily to the 4th and then decreased to a level at the 5th harvest which persisted until the last. Low levels of infection also occurred at the last few harvests in the sand and sandy loam, but in the clay infection rose again at the last two harvests to equal the earlier high levels.

Inoculation increased infection between the first and second harvests in all histories. Figure 25 illustrates that infection was not at its highest at the same harvests in each history: in the 0, 1 and 3 years of cereals histories maximum infection came in the 4th and 5th, 3rd to 5th, and 2nd to 4th harvests, respectively. In the case of the 4- year cereal history, the greatest infection levels occurred in the 2nd to 5th harvests and thus the trend in pattern change was not continued; however it is clear that infection increased earlier in samples with longer cereal histories.

Analysis of variance indicated that over the harvests soils interacted with treatments and histories but that treatments did not interact with histories.

The interaction of soil and history with time is shown in figure 26. The only difference between histories on any soil was in the clay loam where infection in the 3- and 4- year cereal histories was greater than in the 0- and 1- year in the first two harvests, with the result that different patterns emerge. The difference apparent in the 1- year history at the 2nd harvest in the clay does not result in a different pattern.

Figure 27 illustrates that soil influenced infection patterns in the uninoculated treatment. Inoculation after harvest 1 raised overall infection per root as stated previously, but did not effect major changes in pattern compared with treatment 1; see figure 28.

Fig 26

The effect of soil and history on take-all development

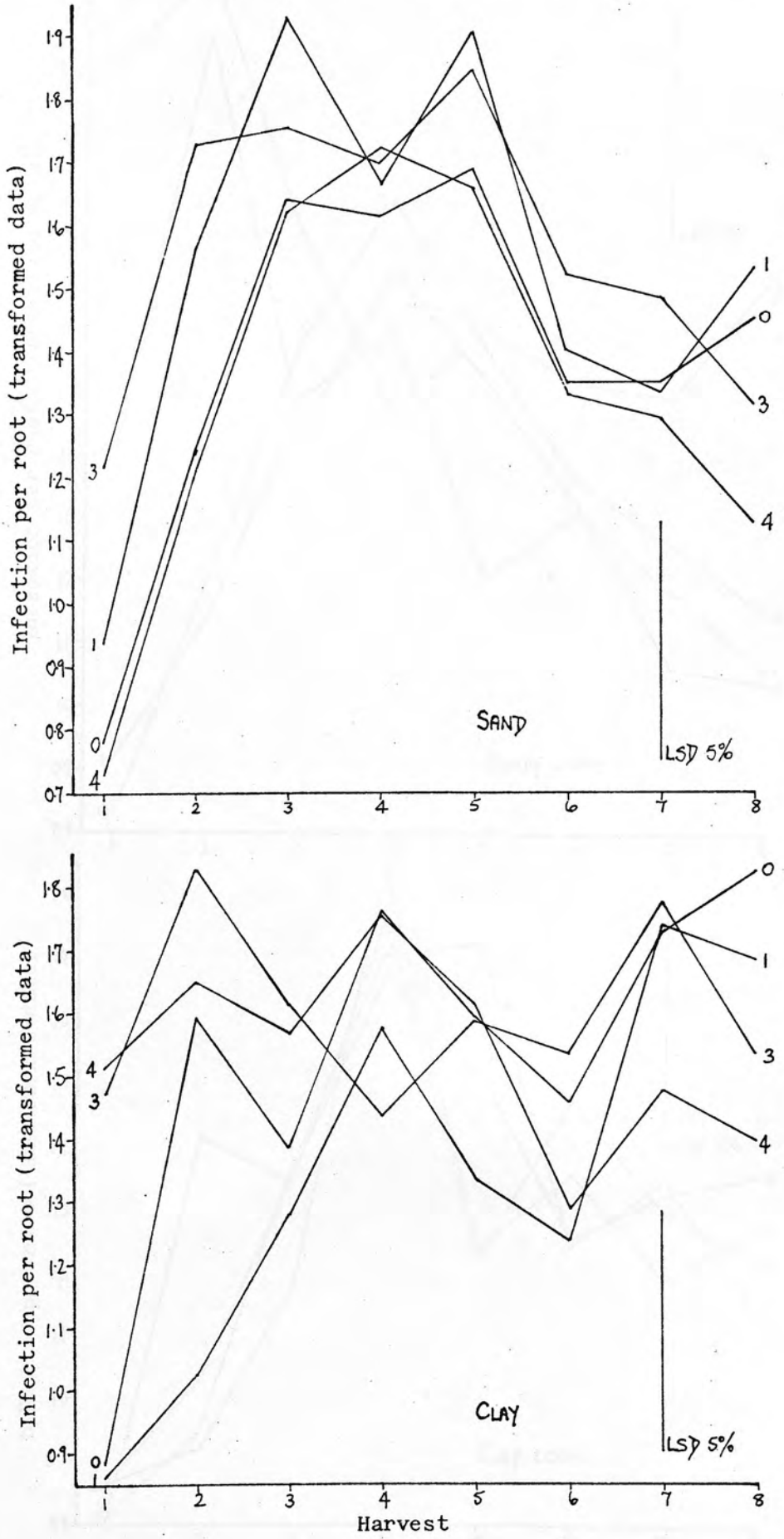


Fig 26 continued

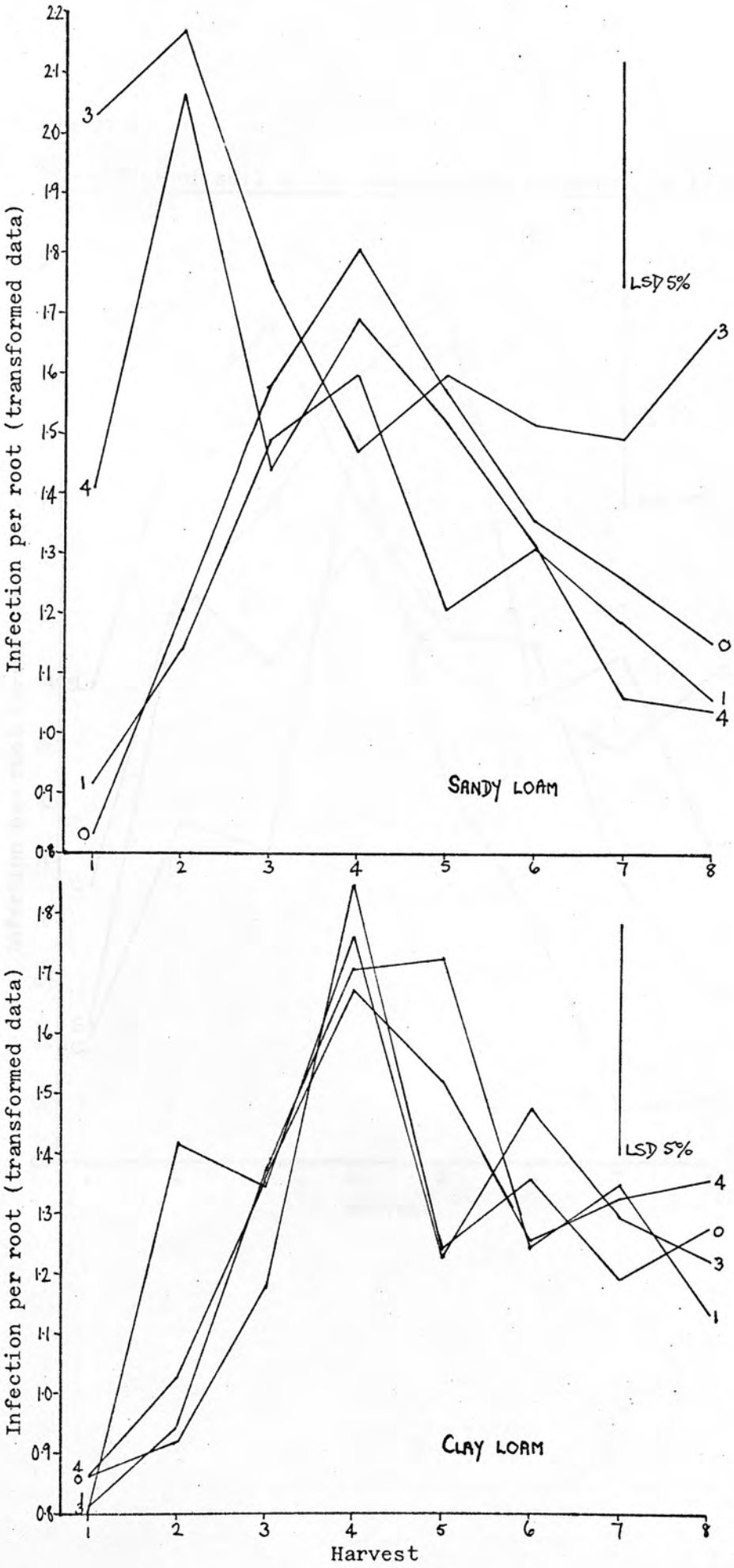


Fig 27

The effect of soil in the uninoculated treatment on take-all development

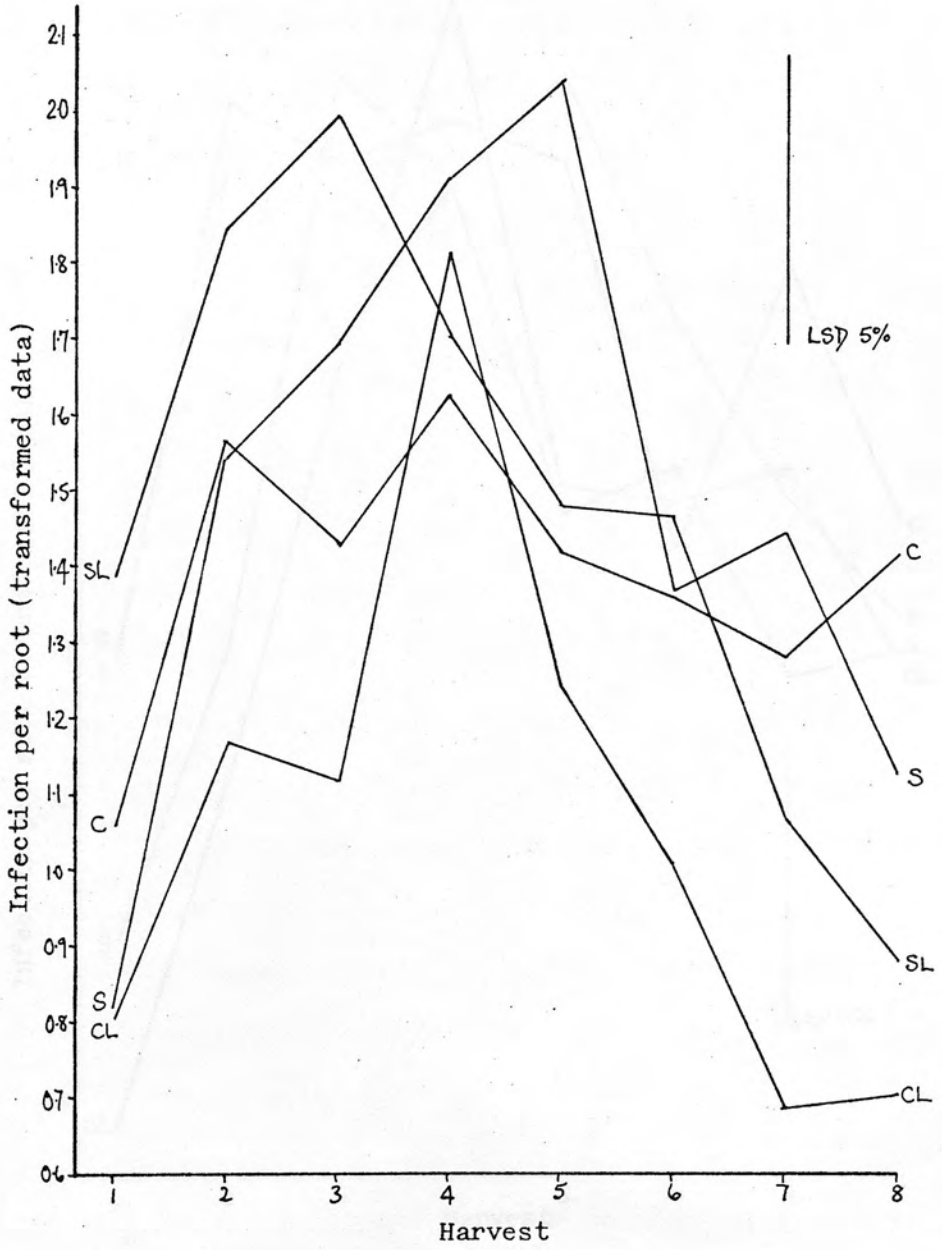
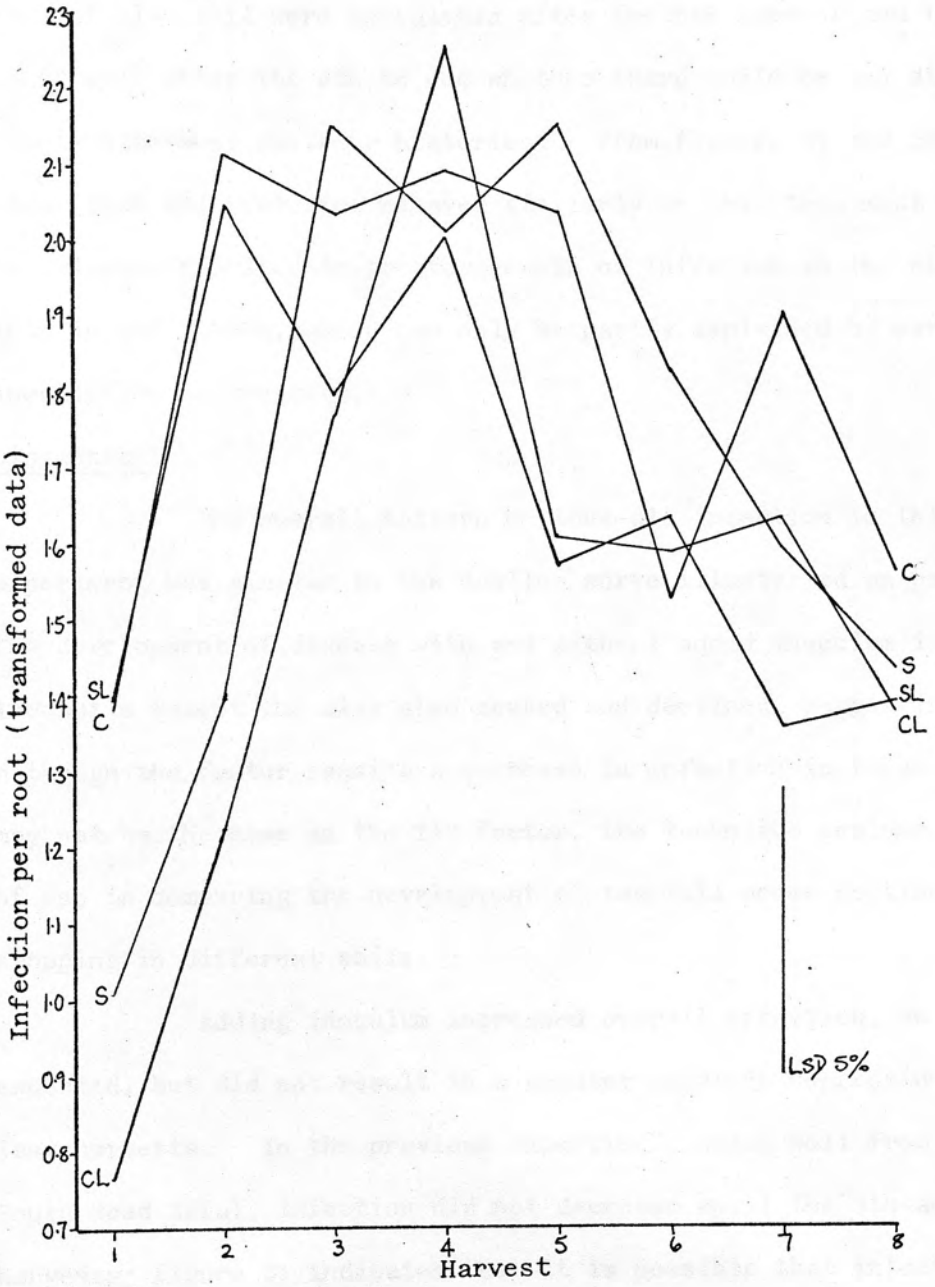


Fig 28

The effect of soil in the inoculated treatment on take-all development



The third treatment: attempted exhaustion of G.graminis with continuous swede cropping, then inoculation as in treatment 2, showed that in no case was the fungus completely eliminated. The pots of clay soil were inoculated after the 5th harvest and those of other soil after the 6th to see whether there would be any differential reaction between soils or histories. From figures 29 and 30 it is clear that all histories behaved similarly in this treatment but that inoculation resulted in greater levels of infection in the clay soil than in all others, which can only be partly explained by earlier inoculation in the clay.

Discussion

The overall pattern of take-all infection in this experiment was similar to the decline curve illustrated on page 4. The development of disease with and without added inoculum in all the soils except the clay also peaked and declined, suggesting that although the factor causing a decrease in infection in these experiments may not be the same as the TAD factor, the technique employed here may be of use in comparing the development of take-all under continuous cereal cropping in different soils.

Adding inoculum increased overall infection, as was expected, but did not result in a greater relative depression in the last harvests. In the previous experiment, using soil from the South Road trial, infection did not decrease until the 9th and 10th harvests; figure 31 indicates that it is possible that infection in the clay soil may have diminished if the experiment had continued for a few more harvests.

The lack of any difference between treatments 2 and 4 indicates that the attempted heat-sterilisation treatment failed to

Fig 29

The effect of soil in the third treatment on take-all development

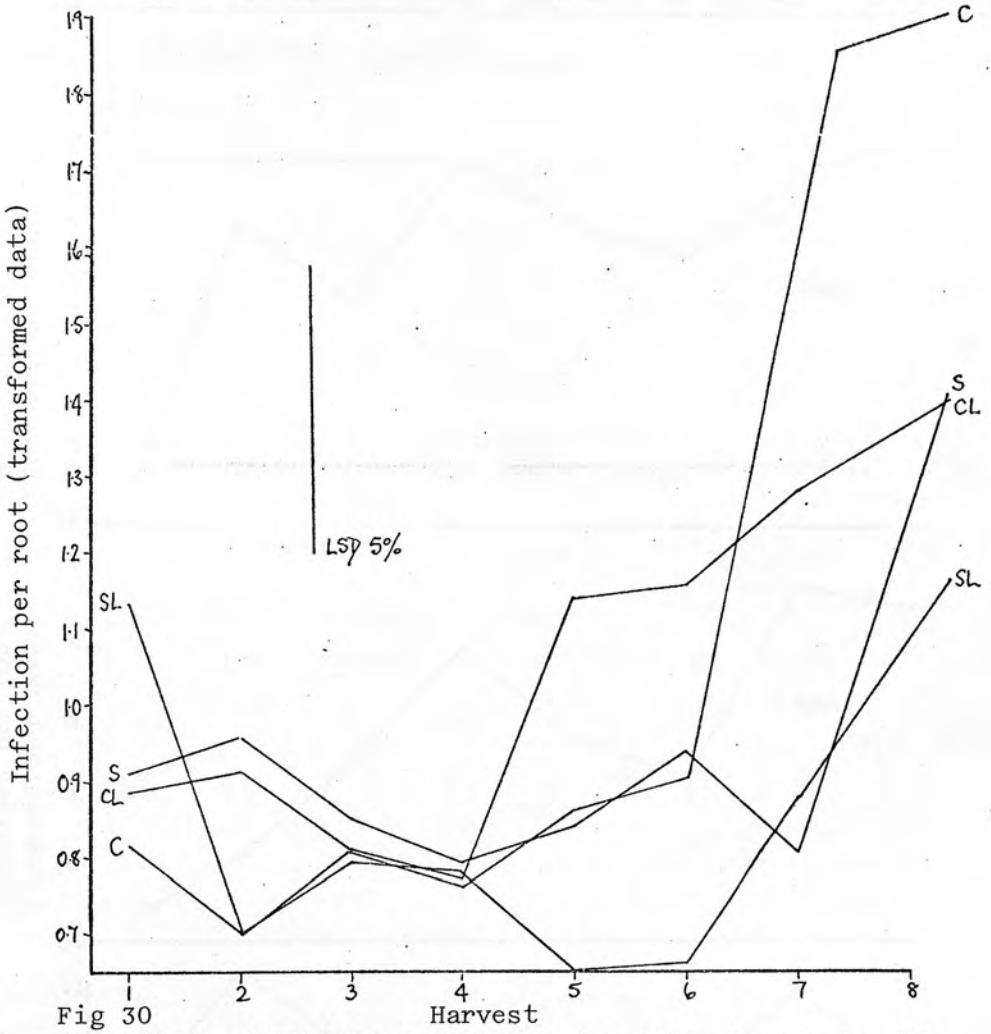


Fig 30

The effect of history in the third treatment on take-all development

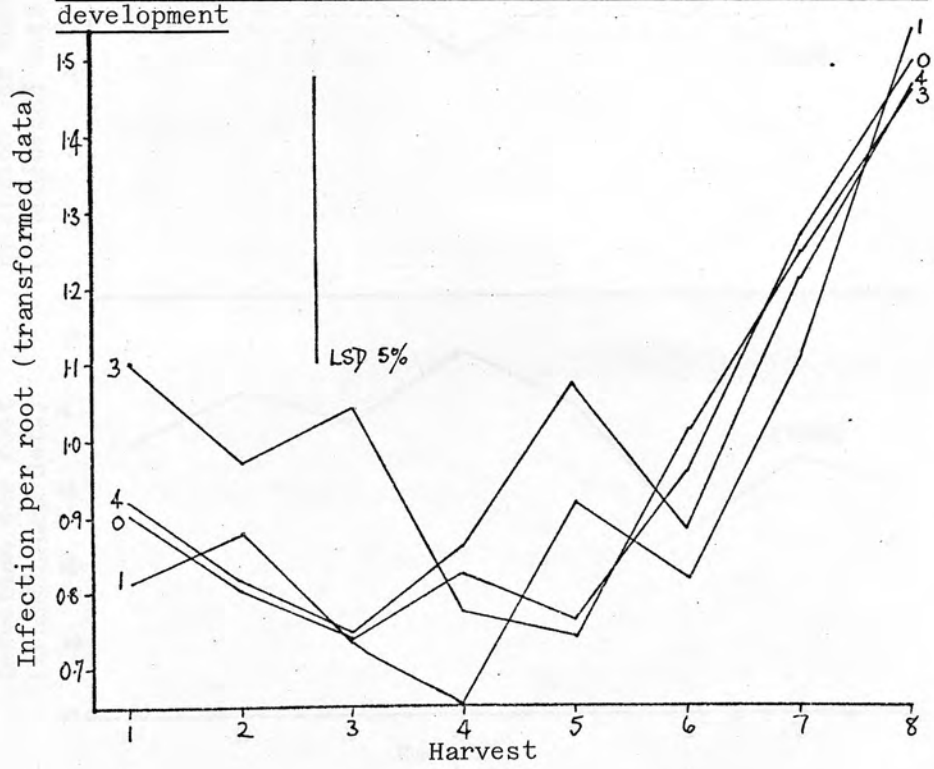
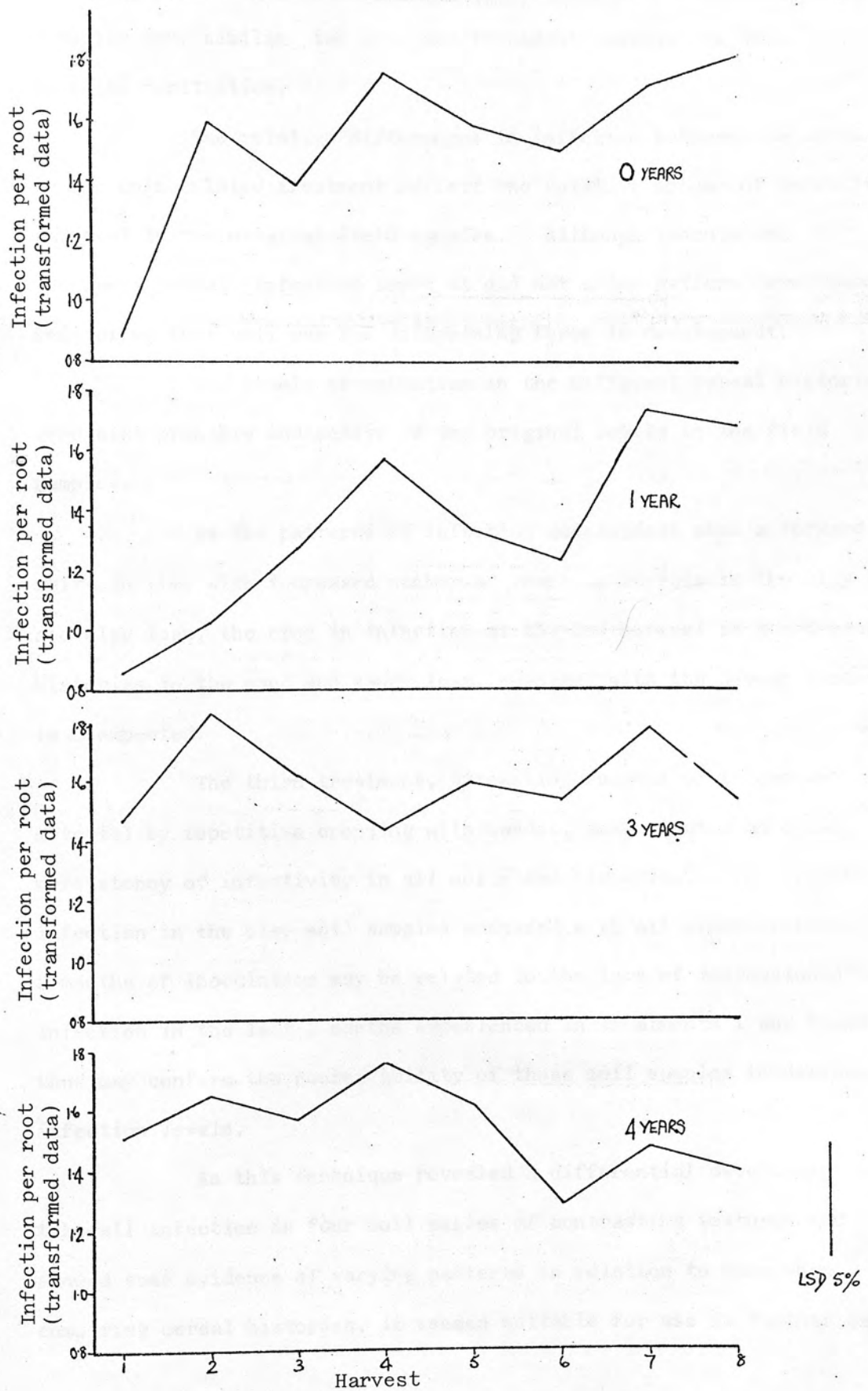


Fig 31

The effect of history in the clay soil on take-all development



remove any competitive or antagonistic micro-organisms and that, since the results were not only insignificantly different, but actually very similar, two pots per treatment combination were adequate replication.

The relative differences in infection between the soils in the uninoculated treatment reflect the relative amount of infective material in the original field samples. Although inoculation increased overall infection level it did not alter pattern development, indicating that soil was the determining force in development.

The levels of infection in the different cereal histories were also probably indicative of the original levels in the field samples.

As the patterns of infection development show a forward shift in time with increased number of years in cereals in the clay and clay loam, the crop in infection at the 2nd harvest of the 4-year histories in the sand and sandy loam, compared with the 3-year history, is unexpected.

The third treatment, attempting removal of infective material by repetitive cropping with swedes, demonstrated an equal persistency of infectivity in all soils and histories. The greater infection in the clay soil samples compared with all others within 2 months of inoculation may be related to the lack of depression of infection in the last 2 months experienced in treatments 1 and 2 and thus may confirm the poorer ability of those soil samples to decrease infection levels.

As this technique revealed a differential development of take-all infection in four soil series of contrasting textures and showed some evidence of varying patterns in relation to time when comparing cereal histories, it seemed suitable for use in further experiments.

EXPERIMENT 3: A COMPARISON OF INFECTION PATTERNS FOUND IN THREE SOIL SERIES, EACH WITH FOUR CROPPING HISTORIES AND SUBJECTED TO A CULTIVATION TREATMENT

The aims of this experiment were to assess the general applicability of the results of Experiment 2 to, eg all soil series of a particular topsoil texture, or even all samples of a particular soil series. It was also thought that more might be found out about the soils and the technique itself by using a greater range of cereal histories. However, fields over 3 or 4 years in cereals were not common in the south east of Scotland, and examples of 0, 4, 8 and 11 or 12 years histories could only be found for the Hobkirk, Macmerry and Winton series. A treatment was imposed to simulate the effects of cultivation and direct drilling using two replicates per treatment for each soil sample and direct drilling.

Materials and methods

The technique employed was the same as that used in the control treatment (treatment 1) of the preceding experiment. In September, 1976 each field sample was collected, mixed thoroughly and divided among 30 350 ml earthenware pots which had been washed and heat-sterilised at 100^{o}C overnight before use. In each pot seven wheat seeds were sown. Of the 30 pots of soil from each field sample, 14 were ear-marked for cultivation treatment before each subsequent sowing. To simulate cultivation the contents of the pot, after removal of the shoots of the previous month's seedlings, was inverted into a bowl, thoroughly mixed by hand and returned to the pot for resowing. The uncultivated pots were resown as described in the basic method.

The pots were arranged in harvest groups and assigned harvest numbers at random. After each replanting the remaining pots were relocated according to a new randomisation.

Results

The general development of take-all in the different soils is shown in figure 32. Differences between the soils occurred only in the last three harvests when the infection in the Hobkirk samples was significantly lower than in the Winton in the 6th, and than in both the Macmerry and Winton samples in the 7th and 8th harvests. However in the 6th and 7th harvests this effect was confined to the cultivation treatment: table 3, shows that, when undisturbed, infection developed similarly in all soils.

TABLE 3: THE EFFECT OF CULTIVATION ON TAKE-ALL INFECTION IN SAMPLES FROM THREE SOIL SERIES (cm infected root per root)

		harvest							
Treatment	Soil series	2	3	4	5	6	7	8	Mean
'uncultivated'	HK	3.5	6.4	4.9	4.0	5.2	4.4	3.2	4.5
	ME	4.1	6.5	5.3	4.8	6.4	6.3	4.9	5.8
	WN	4.7	4.8	5.5	5.5	5.2	5.3	6.0	5.3
'cultivated'	HK	2.5	3.8	4.0	3.3	2.8	2.5	3.3	3.2
	ME	2.4	4.2	4.2	4.1	3.4	3.8	4.2	3.8
	WN	1.9	3.8	4.0	4.2	6.1	5.2	4.4	4.2
SE (difference)		± 0.84	0.90	0.88	0.85	0.80	0.79	0.60	

Cultivation treatment was found to have a major effect throughout the trial, lowering infection significantly in all but the 5th and 8th harvests; see figure 33.

From figure 34 it can be seen that cereal history affected take-all development in the 2nd and 8th harvests. In the 2nd, infection increased less from harvest 1 in the 0-year history than in the other three but in the last harvest infection was greatest in the longest history.

The interaction between cultivation treatment and history is shown in table 4. With the cultivated treatment only, the infection

Fig 32

The effect of soil on development of take-all

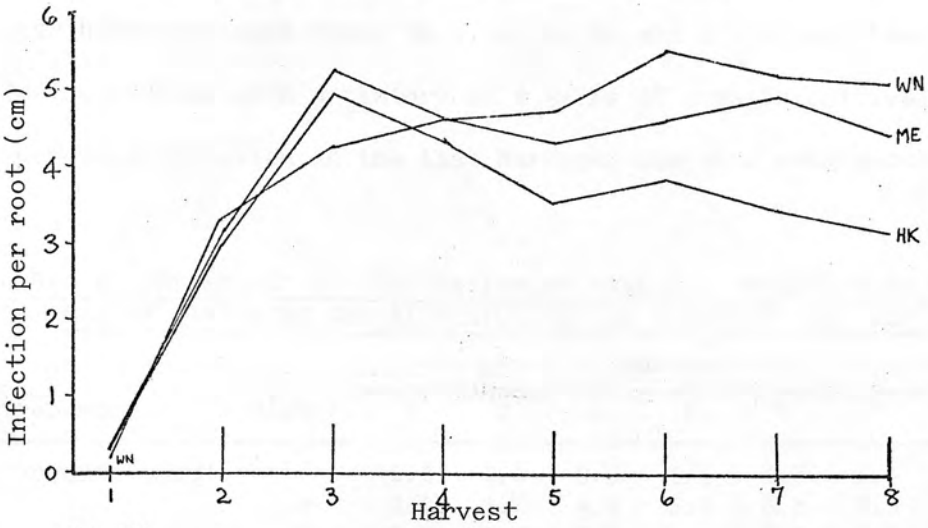


Fig 33

The effect of cultivation on development of take-all

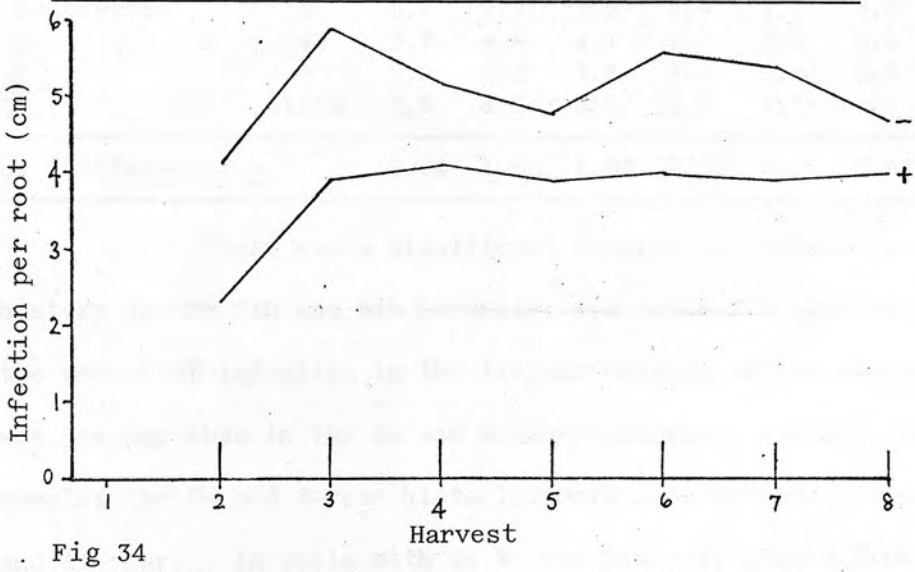
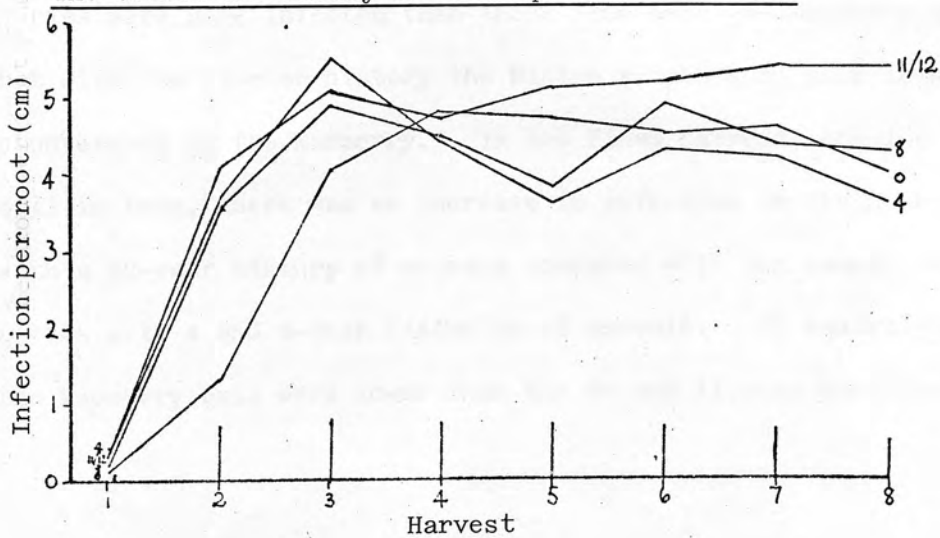


Fig 34

The effect of history on development of take-all



in the 4-year history was significantly lower than in the others at the 5th harvest and at the last harvest the levels in the 0 and 4-year histories were lower than in the 8- and 11/12 year histories. In the samples with a history of 8 years of cereals cultivation increased infection in the last harvest, compared with uncultivated soils.

TABLE 4: THE EFFECT OF CULTIVATION ON TAKE-ALL INFECTION IN SOIL SAMPLES OF DIFFERING CEREAL HISTORIES (cm infected root per root)

		harvest							
Treatment	History	2	3	4	5	6	7	8	Mean
'uncultivated'	0	1.8	5.4	5.0	4.5	4.7	4.6	5.1	4.4
	4	3.6	6.7	4.8	5.8	5.5	5.3	4.6	5.2
	8	5.2	5.6	5.3	3.4	6.3	5.1	3.6	4.9
	11/12	5.8	6.1	5.7	5.4	5.9	6.6	5.5	5.9
'cultivated'	0	0.7	2.9	4.6	4.9	4.4	4.5	3.0	3.6
	4	3.7	4.5	4.1	1.7	3.2	3.2	2.5	3.3
	8	2.2	4.2	3.8	4.1	3.8	3.8	5.1	3.9
	11/12	2.5	4.2	3.8	4.7	4.7	4.2	5.2	4.2
SE (difference) ±		0.98	1.05	1.02	0.98	0.95	0.92	0.70	

There was a significant interaction between soil and history in the 5th and 8th harvests; see table 5. In the 5th harvest the amount of infection in the 11-year history of the Macmerry series was greater than in the 4- and 8-year histories, while in the Winton samples the 0- and 8-year histories were more infected than the 4- and 11-year. In soils with an 8-year history, plants from the Winton series were more infected than those from both the Macmerry and Hobkirk, but with the 11-year history the Winton series soil gave less than its counterpart in the Macmerry. In the final harvest, considering each soil in turn, there was an increase in infection in the Hobkirk samples with a 12-year history of cereals compared with the samples of the same series with 4 and 8-year histories of cereals. In equivalent histories the Macmerry soil were lower than the 0- and 11-year histories but in

the Winton soil infection significantly increased with length of history up to 8 years, and then decreased. In the non-cereal samples the Macmerry series was most infected, but in the 4- and 8-year the levels were highest in the Winton; there was no difference between soils in the longest cereal history.

TABLE 5: THE EFFECT OF THE INTERACTION OF SOIL AND HISTORY ON TAKE-ALL INFECTION (cm infected root per root)

Harvest	Soil	history (years in cereals) 0	4	8	11/12	SE (difference)
1	HK	0.3	0.5	0.4	0.2	
	ME	0.1	0.7	0.2	0.4	± 0.18
	WN	0.1	0.1	0.1	0.5	
2	HK	0.9	3.4	3.4	4.2	
	ME	2.2	3.6	2.6	4.5	± 1.23
	WN	0.3	4.0	4.9	3.7	
3	HK	3.8	5.2	4.8	6.2	
	ME	5.8	6.0	4.9	4.3	± 1.27
	WN	2.0	5.4	5.0	6.7	
4	HK	4.1	4.5	3.8	5.3	
	ME	5.1	4.6	5.1	3.9	± 1.25
	WN	5.3	4.1	4.6	4.8	
5	HK	3.5	3.8	2.8	4.5	
	ME	4.8	3.9	2.4	6.8	± 1.19
	WN	5.8	3.0	6.5	3.8	
6	HK	3.9	4.0	3.3	4.4	
	ME	3.8	3.9	5.6	5.7	± 1.13
	WN	6.0	4.9	6.0	5.7	
7	HK	3.0	3.5	2.8	4.7	
	ME	5.1	5.0	4.1	5.9	± 1.16
	WN	5.4	4.1	6.4	5.5	
8	HK	3.5	2.6	2.0	4.8	
	ME	5.5	3.1	3.8	5.8	± 0.85
	WN	3.1	5.0	7.3	5.5	
Mean		3.5	3.7	3.8	4.4	

Cultivation affected the soil-history interaction at the 6th and 8th harvests. In the 6th harvest, infection decreased with cultivation in the Hobkirk 8 and Macmerry 4 and 8 samples but was increased in the Winton 0 sample; in the last harvest cultivation

increased infection in the Winton 8 sample but decreased it in Hobkirk 4 and the 0-year histories of Macmerry and Winton. Table 6 records the effect of cultivation.

TABLE 6: THE EFFECT OF CULTIVATION TREATMENT, SOIL SERIES AND CEREAL HISTORY ON TAKE-ALL INFECTION IN HARVESTS 6 AND 8 (cm infected root per root)

Harvest 6

Treatment	Soil	History (years) 0	4	8	11/12
'uncultivated'	HK	5.7	4.2	5.6	6.2
	ME	5.7	6.8	7.9	5.3
	WN	3.6	5.4	5.5	6.4
'cultivated'	HK	3.1	3.8	1.5	2.9
	ME	2.3	1.4	3.8	6.0
	WN	8.4	4.3	6.5	5.3

SE (difference) ± 1.72

Harvest 8

'uncultivated'	HK	2.4	4.0	2.1	4.2
	ME	7.2	3.8	2.7	5.9
	WN	5.6	6.1	5.9	6.4
'cultivated'	HK	4.5	1.2	1.8	5.4
	ME	3.9	2.5	4.8	5.7
	WN	0.5	3.9	8.7	4.5

SE (difference) ± 1.21

In the uncultivated treatment there was no interaction between soils and histories in the 6th harvest but in the last the 4- and 8-year histories had less infection than the 0- and 11-year in the Macmerry samples in the cultivated treatment the interaction was quite different: at the 6th harvest levels tended to be highest in soil of the 11-year cereal history in the Macmerry series whereas in the Winton soil the 0-year history gave the highest infection. In contrast, at the last harvest the Winton 0 samples had very little infection and high infection occurred in Winton 8. In the Macmerry series the 11-year cereal history was most infected with significantly

more disease than Macmerry 4. In the Hobkirk samples the longest cereal history was significantly more infected than Hobkirk 4 and 8.

The data of this experiment are tabulated in Appendix II.

Discussion

The disease response of the three soils was not similar to that of samples in the two earlier experiments: although infection was found to have increased at the second harvest no peak and decline shape was discernible and the pattern did not occur progressively earlier with increasing number of years in cereals, as found, for example, in the clay soil in experiment 2; see figure 31. The mean infection level, 3.9 cm infected root/root is similar to the mean level of the South Road samples used in experiment 1: 3.1 cm, it thus seems that no general suppressive agent was active.

The effect of cultivation treatment in reducing infection was unexpected, as take-all infectivity is usually greater in cultivated soil than in direct-drilled conditions. However an interaction of cultivation treatment with history is not unprecedented: in the South Road trial the relationship of infection in the unploughed plots compared with the ploughed changed with time, particularly at the early assessments; see figures 2 and 3. The technique used to simulate cultivation may have been responsible for the reduction, as inoculum may have remained in a critical zone for subsequent infection in the undisturbed pots but have been dispersed less favourably for infection during the physical breaking and repacking of the soil ball in the disturbed treatment.

EXPERIMENT 4: A COMPARISON OF TAKE-ALL DEVELOPMENT IN MISCELLANEOUS SOIL SAMPLES COLLECTED IN 1975 AND 1976.

This experiment compared the development of infection in samples of a wide range of soil series to gain information about soils on which as yet only short periods of continuous cereals had been grown. It was intended that this would simulate the assay when in use as an advisory tool.

Materials and methods

Group a: In 1975 soil samples from 35 fields representing 14 soil series in the cereal-growing areas of East Lothian and the eastern Borders Region of Scotland were collected and assayed according to the basic method described on page 78 . No treatments were imposed and no inoculum was added.

Group b: In 1976 soil samples were collected from 15 fields not currently growing a cereal crop and were assayed in the same manner as Group a.

Two pots of soil from each field sample were assayed at every harvest. The pots were arranged in harvest groups and assigned harvest numbers at random. After each replanting the remaining pots were relocated according to a new randomisation.

Results

In Group a there were six Winton samples and four Biel series samples; their infection development is shown in figures 35 and 36. The assay seedlings of harvest 6 were accidently discarded before assessment.

The mean infection level in the Winton assays was 2.8 cm infected root/root and the overall development pattern shows a peak at the third harvest followed by a decrease. The samples from a field

Fig 35

The development of take-all in Winton soil samples of different histories

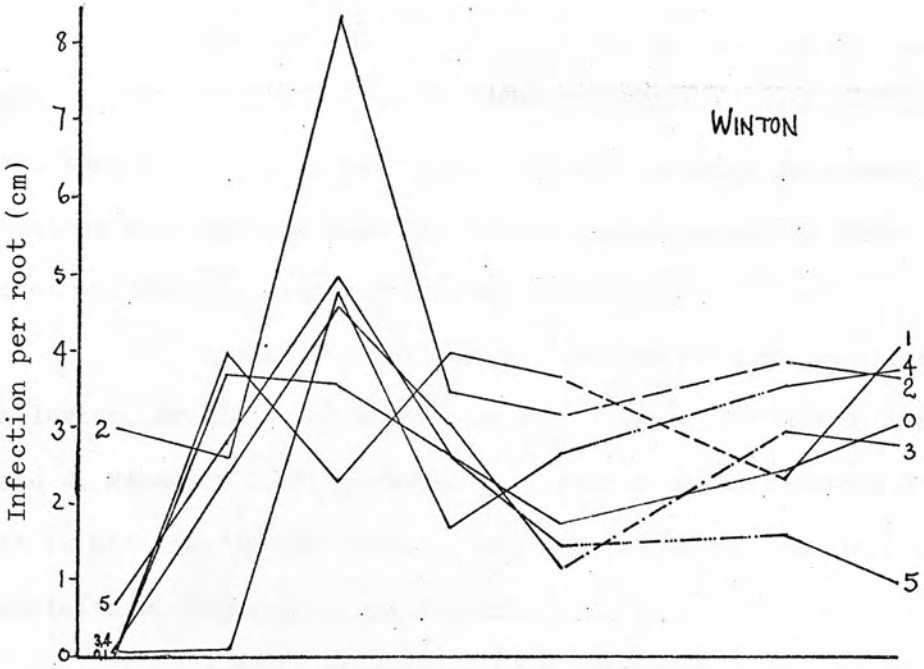
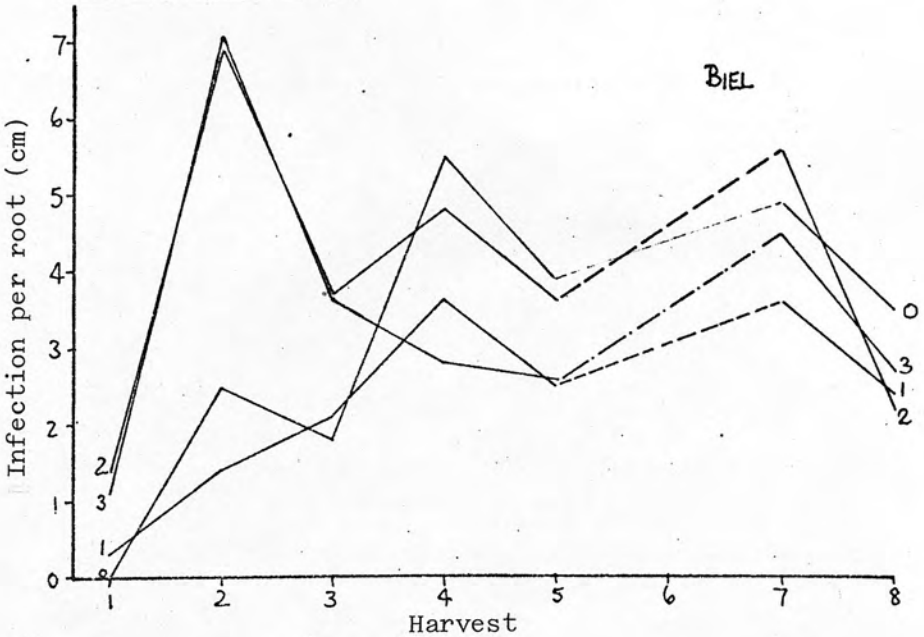


Fig 36

The development of take-all in Biel soil samples of different histories



with a 2-year history of cereal cropping were relatively highly infected compared with the others which were similar to the mean. The maximum level of infection in each history usually occurred at the third harvest, and there was no evidence of an advancement of the peak with increasing years in cereals. A sustained drop in level of infection after the peak only occurred in the 2- and 5-year cereal history samples.

The pattern of infection in the Biel series, averaged over all samples, revealed an irregular development: although infection in the samples of the 2- and 3-year cereal histories increased to a maximum more quickly than the 0- and 1-year histories there was no clear evidence of subsequent stable decline.

A peak and decline pattern can be seen in all the Alluvium, Dreghorn and Morham samples (Figure 37) and in the Darleith 2 and 4, Macmerry 1, Fraserburgh 2, Peffer 3 and Kilmarnock 5 samples. It is not seen in the Yarrow, Cessford and Belses samples, nor in Darleith 0, Macmerry 3 and Hobkirk 0.

Table 7 shows that mean infection level was highest in the clay loam samples and tended to increase with increasing clay content.

Fig 37

The development of take-all in miscellaneous soil samples - Group a

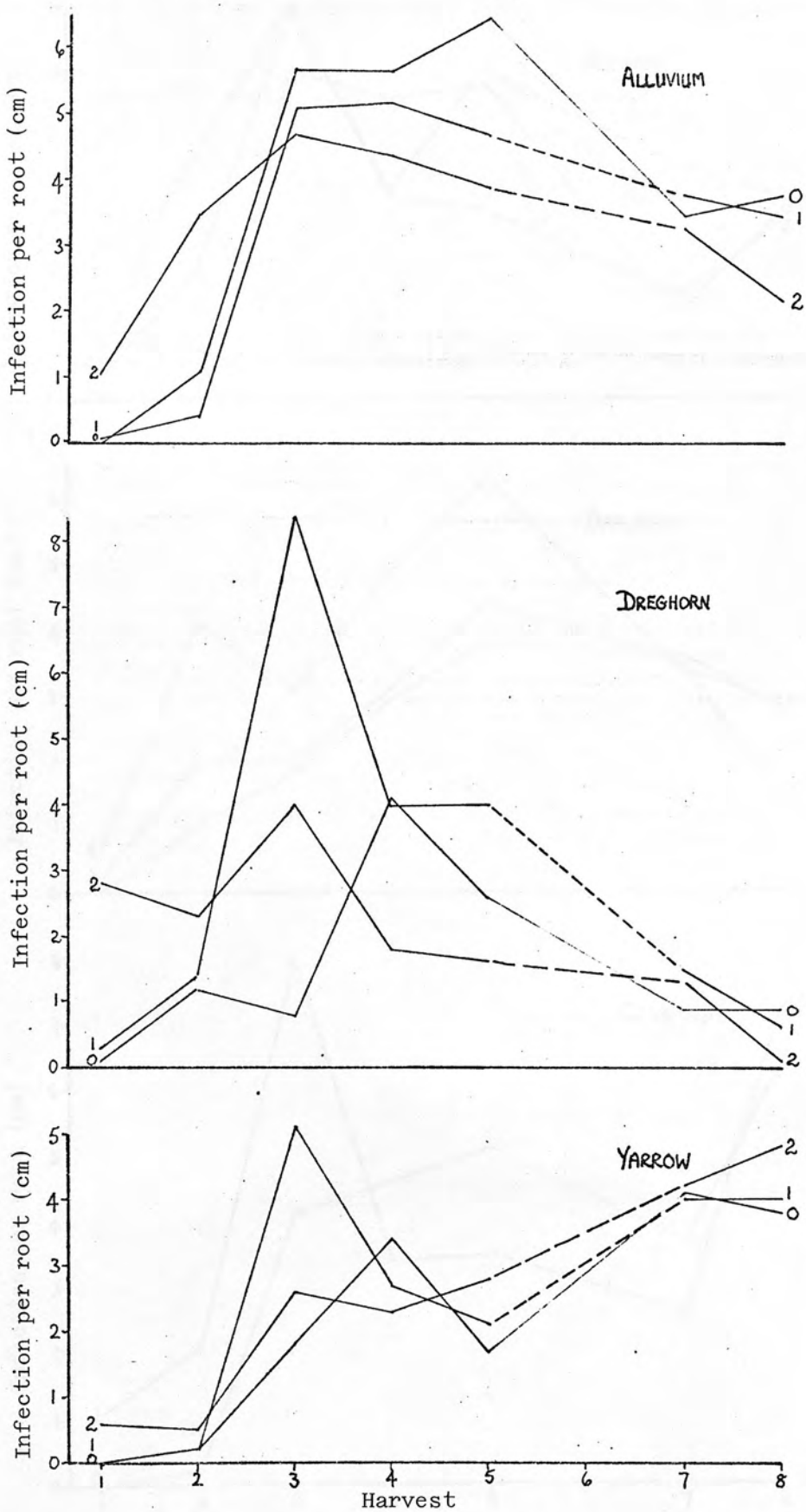


Fig 37 continued

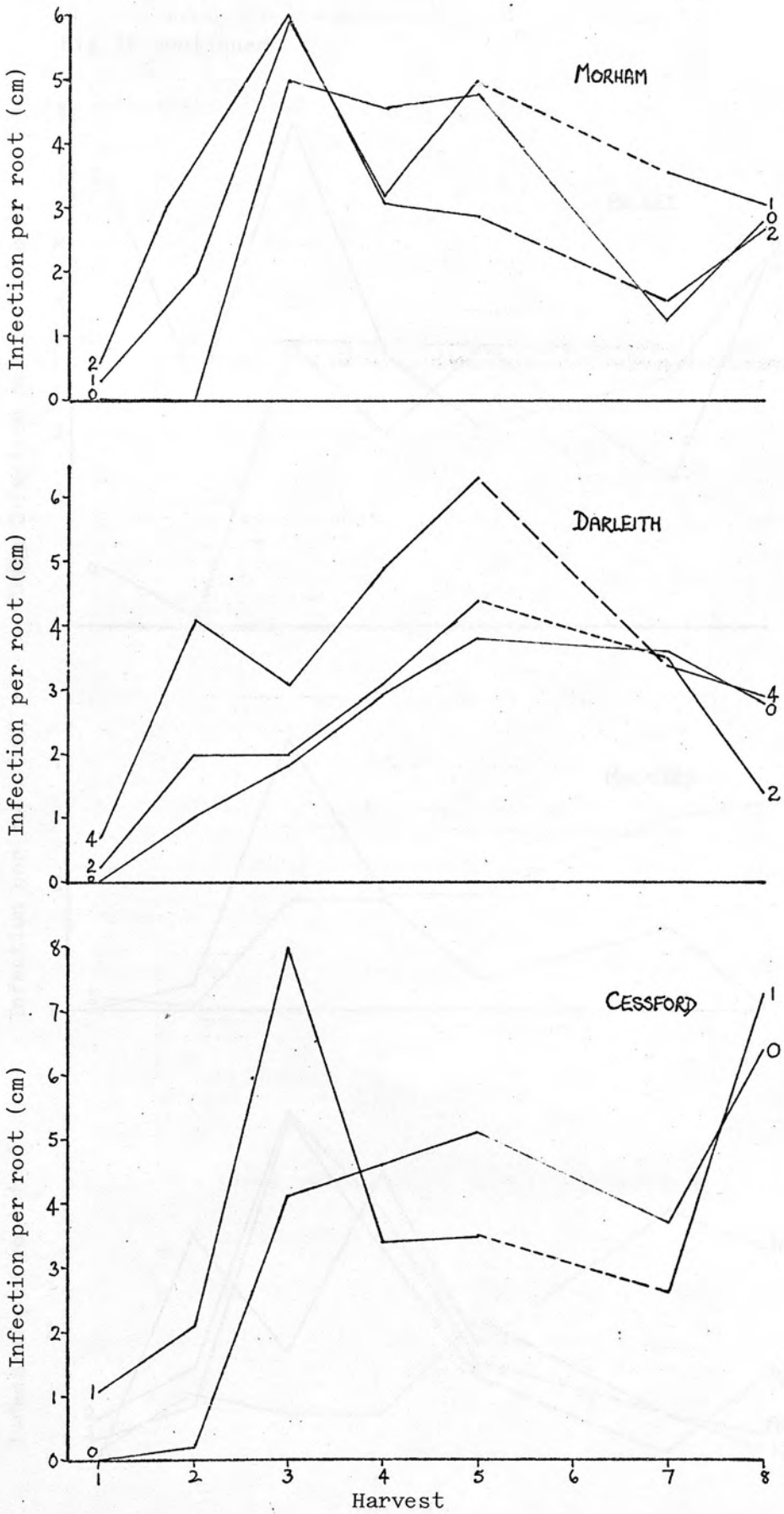


Fig 37 continued

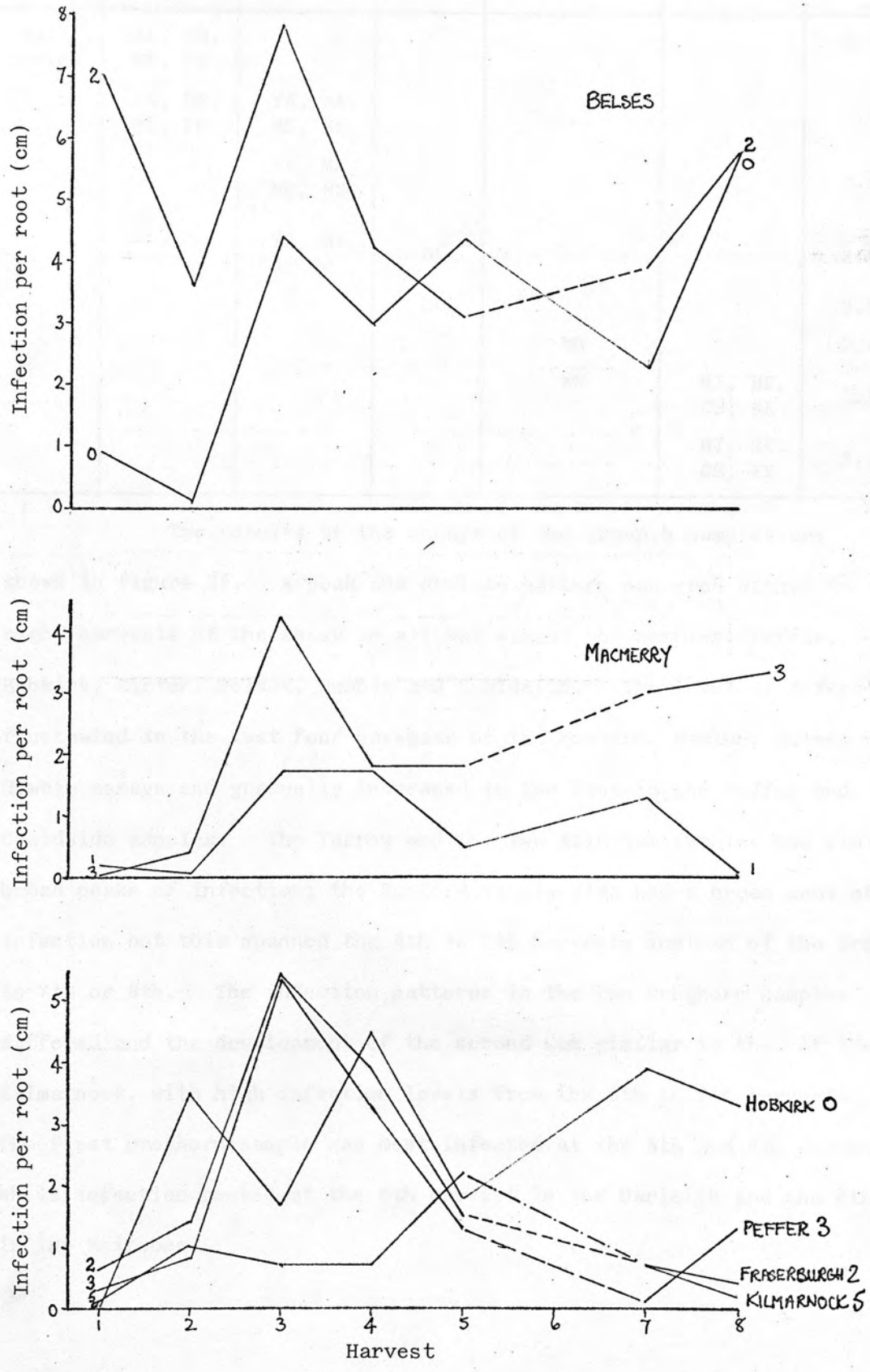


Table 7: the effect of soil texture on the mean level of take-all infection (cm infected root per root)

	soil texture					
	Sand	Sandy loam	Loam	Sandy clay loam	Clay loam	Mean
Soil series	AL, DR, PF, FR					2.5
	AL, DR, PF, FR	YA, MA, ME, HK				2.5
		YA, MA, ME, HK				2.4
		YA, MA, ME,HK	DL			2.5
			DL			2.8
				WN		2.8
				WN	BI, BE, CS, KK	3.1
					BI, BE, CS, KK	3.2

The results of the assays of the group b samples are shown in figure 38. A peak and decline pattern occurred within the eight harvests of the assay in all but six of the samples: Peffer, Hobkirk, Winton, Belses, Humbie and Cauldside. The level of infection fluctuated in the last four harvests of the Hobkirk, Winton, Belses and Humbie assays and gradually increased to the last in the Peffer and Cauldside samples. The Yarrow and the two Alluvium samples had similar broad peaks of infection; the Eckford sample also had a broad peak of infection but this spanned the 4th to 7th harvests instead of the 3rd to 7th or 8th. The infection patterns in the two Dreghorn samples differed and the development of the second was similar to that of the Kilmarnock, with high infection levels from the 5th to 7th harvests. The first Dreghorn sample was most infected at the 5th and 6th harvests while infection peaked at the 5th harvest in the Darleith and the 6th in the Whitsome.

Fig 38

The development of take-all in miscellaneous soil samples - Group b

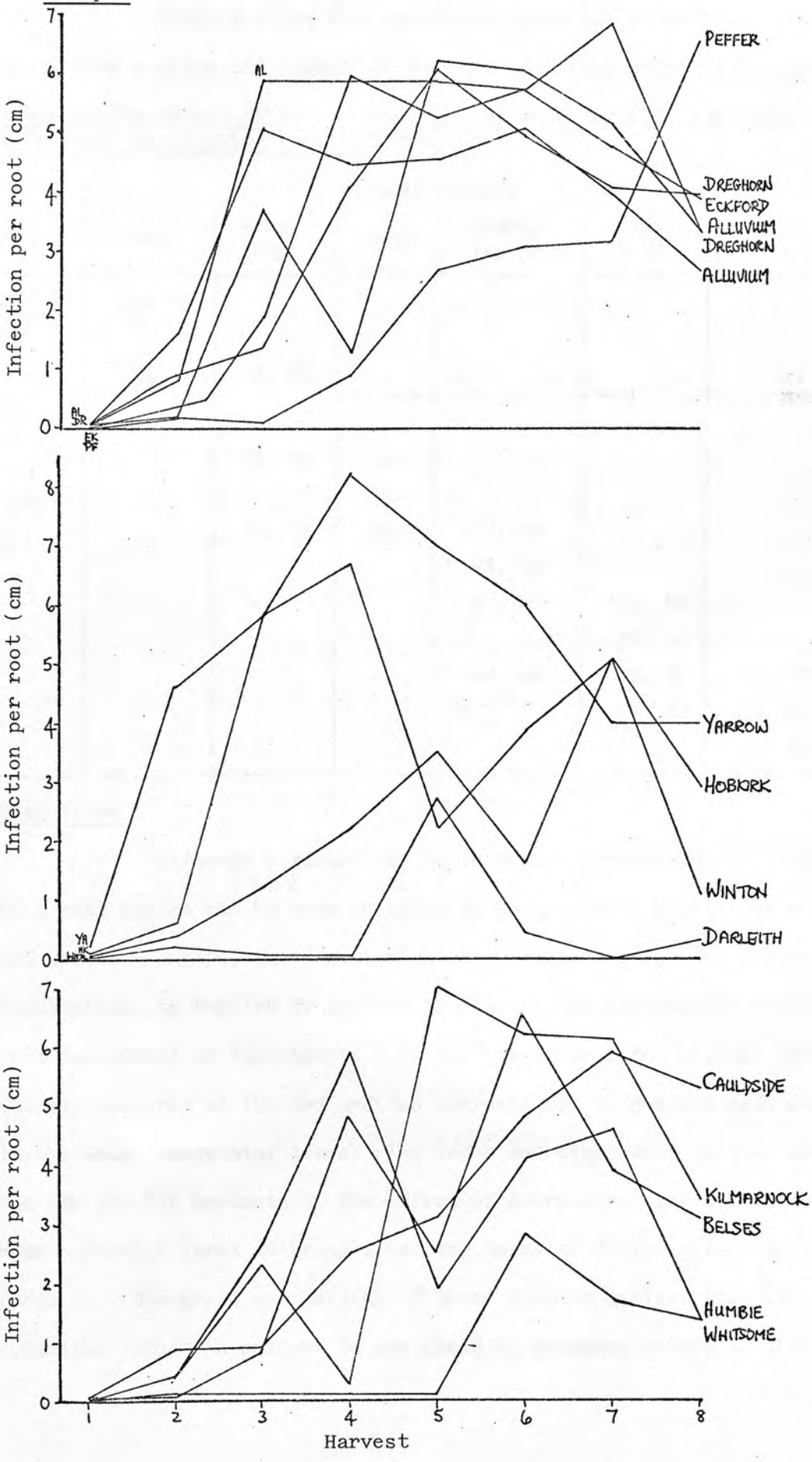


Table 8 shows that infection level was highest in the sandy loam samples and tended to decrease with increasing clay content.

Table 8: the effect of soil texture on the mean level of take-all infection (cm infected root per root)

	Soil texture						
	Sand	Sandy loam	Loam	Sandy clay loam	Clay loam	Clay	Mean
Soil series	AL, DR, EK, PF						3.0
	AL, DR, EK, PF	YA, HK					3.5
		YA, HK					4.2
		YA, HK	DL				3.0
			DL				0.5
		YA, HK	DL	HM, WN			2.7
				HM, WN			2.3
				HM, WN	KK, BE		2.6
					KK, BE		2.9
				HM, WN	KK, BE	CU, WH	2.3
					KK, BE	CU, WH	2.4
						CU, WH	1.8

Discussion

Although similarities in infection pattern between histories of a soil series can be seen in group a, particularly within the Winton and Alluvium assays, the development of infection in any soil/history combination, eg Hobkirk 0, was not similar to its equivalent sample in this experiment or Experiments 2 or 3. In group a the highest infections usually occurred at the 2nd and 3rd harvests but in group b most maxima in the loam, sandy clay loams, clay loams and clays were delayed until the 4th and 5th harvests. The effect of increasing clay content on mean infection level in group a was the opposite of its effect in group b. The great variability of these results implies that the potential infection pattern in any field is probably unique. When the

geographical, historical and climatological factors influencing any field pattern are considered this conclusion is not unexpected. It does seem, however, that the peak and decline pattern occurs more often in the sands and sandy loam soils assays than in those of heavier texture.

It was found that attempting to provide stable environmental conditions over the assay period of 8 months was not very easy: probably the greatest variation occurred in temperature as the glasshouse often overheated in summer and in the winter seedlings received direct heat from supplementary lighting. The controlled conditions of a growth cabinet or room would therefore have been advantageous.

At the end of the first experiment in this chapter the question of the relationship of the patterns of infection found during assay and the potential development of take-all in the field was raised. To obtain a full answer long-term cereal trials would have to be set up on a number of soils. Although the data for the assay of the Macmerry soil of the South Road trial are incomplete, they describe an ultimate decrease in infection in the 9th and 10th harvests; in the field the decrease occurred in the 12th year. It would therefore seem that the period of assay should be extended to 12 months.

Any attempted assay technique would fail to take into account many factors, both environmental and agronomic, which exert an influence on the disease in field conditions. The results of the assays described in this chapter revealed many different patterns of infection and distinguished statistically between some samples but the relationship between these results and the field patterns remains to be demonstrated.

CHAPTER 3

VIRULENCE EXPERIMENTS

Introduction

Virulence is defined in the context of these investigations as the relative capacity of an isolate to cause disease: a more virulent isolate will give rise to a more severe effect on the host following infection.

The assessment of the relative virulence of different populations of G. graminis var tritici is the final part of this study of the factors involved in forecasting take-all disease patterns in long-term cereals. Having investigated the reaction of different soil types of different histories with standard inoculum it remained to take populations of isolates from different soils and histories and assay them in standard media and conditions.

In 1973 Pearson and Hornby declared that there was no satisfactory standard test for pathogenicity (ie virulence) and used an index compounded from measures of numbers of lesions per root and percentage incidence, amount of root length infected and numbers of plants showing shoot damage. As the virulence of an isolate may be reflected in its interactions with its environment, experiments have attempted to quantify, and relate virulence with, certain characteristics such as cellulolytic ability (Pearson, 1973 and 1974), the ability to produce ascospores (Chambers and Flentje, 1967 a and b) and inclusion of virus-like particles (VLP) (Lapierre, Lemaire, Jouan and Molin, 1970; Rawlinson, Hornby, Pearson and Carpenter, 1973). Although Garrett (1975) found that straw-penetration rate in vitro did vary with cellulolysis rate, Holden (1976) was not satisfied that it was the primary determinant of virulence. Abbott and Holland (1975) failed to characterise different isolates by their electrophoretic patterns of soluble proteins

and isoenzymes and Weste (1970 a and b) similarly found that differences in extracellular enzymes were only sensitive enough to distinguish between varieties of G. graminis.

There is an obvious need for a simple, non-labour intensive test for the assessment of virulence of a population of isolates for use in advisory work. Many techniques are impractical for assessing a field population, including the eosin stain uptake method devised by Deacon and Henry (1978) which measures the ability of an isolate to block the vascular tissue of the host. However, their work, and that of Holden (1976), indicate the importance of vascular discoloration of the root upwards from the point of inoculum, and that shoot growth may not always directly reflect events in the root.

When using the technique for producing inoculum described in Chapter 2, it was realised that the technique could be adapted to measure the ability of isolates to grow up host roots. As, theoretically the ultimate test of the potential of an isolate to cause damage to a plant is related to its ability to block vascular tissue, thereby resulting in discoloration, grow up the host root towards the crown, and thus colonise further roots, this basic technique was selected. If a layer of washed, surface-sterilised infected roots is placed at a standard distance from wheat and barley seeds in a pot of washed, heat-sterilised sand, the roots of the seedlings will reach the layer of inoculum at more or less the same time. Subsequent growth of the fungus up the roots should give a measure of potential virulence, irrespective of the possible interactions of different isolates with nutrients (Weste and Thrower, 1971), temperature and pH (Davis, 1925), amino acids (Siegle, 1961;

Timonin, Peterson and Rouatt, 1974), light (Wilkinson, 1970), 'volatiles' from decomposing plant tissues (Lewis and Papavizas, 1974), the effect of suppressive factors in the soil (Pope and Jackson, 1973), and the age of the plant (Broadfoot, 1933a; Deacon and Henry, 1978). This technique ensures that the population is assayed in a parasitic state and has not been required to grow saprophytically, for example on agar, when different enzymes are in use.

No effort was made to assess non-pathogenic isolates, ie isolates producing only runner-hyphae and cortical infections, the importance of which have been discussed by Tivoli, Lemaire and Jouan (1974) and Asher (1978 a and b).

Six experiments were devised to ascertain the possible relationship between the following factors and the virulence of populations of G. graminis:-

1 soil texture

2 soil texture and cereal history

3 cereal rotation and nitrogen level

4 cereal host

5 reassaying

6 previous growth on potato dextrose agar.

Materials and methods

Roots from plant samples were divided into equal lots and placed in a layer about 2 cm from the base of two 350 ml earthenware pots and covered with sand; in each pot seven pre-germinated seeds of wheat, cv Mega, untreated with fungicide, were planted about 8.5 cm above the inoculum and grown in a glasshouse in the same conditions as the experiments described in chapter 2, page 78.

The inoculum and wheat seeds were washed and surface-sterilised with 10 per cent Deosan (hypochlorite) for 5 minutes before adding to the pots; the pots and the sand were washed and heat-sterilised at 100°C overnight before use. Pots were completely randomised and laid out in the sunken bed, as described in chapter 2.

After 28 days the seedlings were washed from the sand; root growth below the layer of inoculum was cut off and the inoculum discarded. The assay seedlings were floated in water and the maximum distance pigmented hyphae had grown up each root from the inoculum layer was measured to the nearest millimetre without the aid of a microscope or staining. The number of uninfected roots in each assay was also recorded.

Experiment 1: effects of soil texture. Infected roots of twenty-eight days old wheat seedlings previously grown in soil samples of different textures were used as inocula in the first group of assays; all came from harvest 6 of the second miscellaneous soils experiment in chapter 2 and are natural inocula. For the second group of assays inocula were the roots of infected plants from cereal crops representing a range of soil textures and histories; they were collected during the summer of 1976.

Experiment 2: effects of soil texture and cereal history. Infected roots of seedlings from the 6th harvest of experiment 3 in chapter 2 were used as inocula. The original samples for the experiment in chapter 2 were collected from twelve fields representing 0, 4, 8 and 11 or 12 years of continuously grown cereals on the Hobkirk, Winton and Macmerry soil series. The treatments 'cultivated' and 'non-cultivated' refer to the soil being disturbed or not between replantings in the original experiment.

Experiment 3: effects of cereal rotation and nitrogen level. For this experiment inoculum was the roots of infected plants from the 1976 harvest of a cereal rotation and nitrogen level trial at North Belton Farm, Dunbar, East Lothian. The rotation treatments were:

A - continuous winter wheat with chemical couch control by glyphosate;

B - continuous winter wheat with mechanical couch control by chisel ploughing;

C - winter wheat (in 1976) rotating with spring barley with no couch control;

D - continuous spring barley with no couch control.

Two of the applied nitrogen treatments were selected for this assay: low and high, being respectively 105 and 210 kg N/ha on wheat and 75 and 180 kg N/ha on barley. There were three replicates. The soil was of the Biel series, being a uniform sandy clay loam over a clay loam subsoil. In 1976 the field was in its 12th cereal crop and the trial was in its 4th year.

Experiment 4: effects of cereal host. Infected roots of seedlings from the second harvest of experiments 3 and 4 in chapter 2 were used as inocula; each batch of inoculum was divided into four lots, two of which were assayed with wheat and two with barley.

Experiment 5: effects of reassaying. In this experiment the assay seedlings from virulence experiment 1a and part of experiment 2 were used as inoculum. Each infected portion of root was divided into 0.5 cm lengths to provide inoculum for one pot.

Experiment 6: effects of previous growth on potato dextrose agar. In the final experiment five infected roots were selected from the Dreghorn assay seedlings in virulence experiment 1a.

After surface sterilisation with 10% Deosan for five minutes the infected portions of the roots were divided into two pieces, where possible, and plated on potato dextrose agar with streptomycin. After seven days four 2 mm^2 pieces of agar from the edge of each colony were transferred to new plates and when the subcultures, A, B, C and D, had grown to a diameter of approximately 6 cm, each was divided equally to form a layer of inoculum for duplicate assays. In some cases, however, contamination of plates resulted in the loss of whole or part of some subcultures.

In the first four experiments the data were divided into strata and subjected to chi^2 analysis. The strata represented infection categories, the bounds of which were changed with each experiment to exploit the greatest sensitivity of the analytical technique. It was found necessary to pool the data from both pots throughout these experiments to provide adequate numbers of infections for analysis. It was not possible to statistically analyse the data from experiments 5 and 6.

Results

A stratified distribution of observed and expected values for the first four experiments are given in Appendix III.

Experiment 1: the effect of soil texture.

The frequency distribution of lengths of infected roots from the assays of populations of isolates from different soil textures is shown in figures 39 a and b and tables 9 a and b.

Fig 39 a

The distribution of lengths of infected roots in the assays of G. graminis populations from miscellaneous soils of non-cereal history

Each millimetre mark represents one infected root.

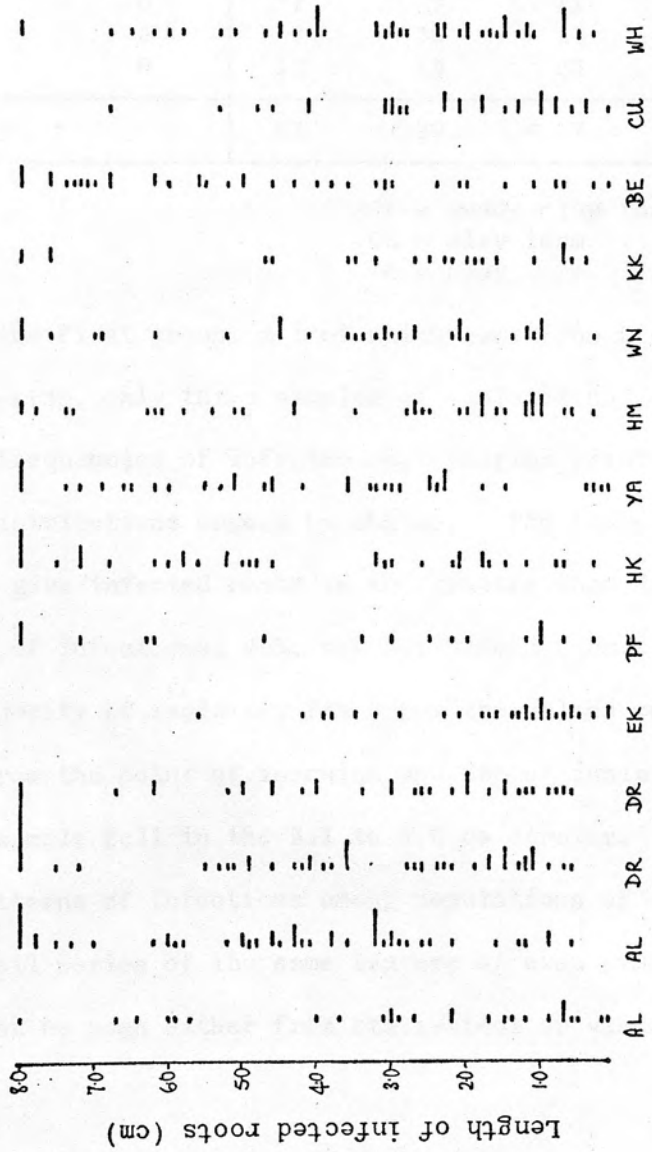


TABLE 9 a: THE EFFECT OF SOIL TEXTURE ON THE DISTRIBUTION OF INFECTIONS IN ASSAYS (PERCENTAGE FREQUENCY OF OBSERVED VALUES)

Soil series	Topsoil texture	Years in cereal	Infection category 0.1-1.0	1.1-2.0	2.1-3.0	3.1-6.0	> 6.0 cm
Hobkirk	SL	0	9	16	13	41	22
Winton	SCL	0	3	22	25	38	13
Kilmarnock	CL	0	24	19	24	24	10
Peffer	S	0	13	27	20	20	20
Cauldside	C	0	19	26	23	29	3
Humbie	SCL	0	8	36	19	19	17
Eckford	S	0	31	38	10	21	0
Whitsome	C	0	13	21	19	38	9
Yarrow	SL	0	12	6	21	41	21
Belses	CL	0	11	9	7	42	32
Alluvium	S	0	31	15	15	27	12
		0	7	7	11	58	18
Dreghorn	S	0	7	30	15	42	5
		0	13	19	23	35	10
Mean			13	20	17	36	14

S = sand
SL = sandy loam
L = loam
SCL = sandy clay loam
CL = clay loam
C = clay

In the first group, all of which came from fields not growing a cereal crop, only three samples of isolates had differences in distribution frequencies of infected root lengths greater than differences in distributions caused by chance. The Eckford series isolates did not give infected roots in the greater than 6 cm stratum and the majority of infections, 69%, did not exceed 2 cm. In contrast, the majority of isolates, 74%, from the Belses series, grew more than 3 cm from the point of inoculum and 58% of isolates from the second Alluvium sample fell in the 3.1 to 6.0 cm stratum. Any similarity in patterns of infections among populations of isolates from different soil series of the same texture or even samples of the same series cannot be seen either from statistical or visual assessment of the data.

Fig 39 b

The distribution of lengths of infected roots in the assays of G. graminis populations from miscellaneous soils of mixed cereal histories

Each millimetre mark represents one infected root.

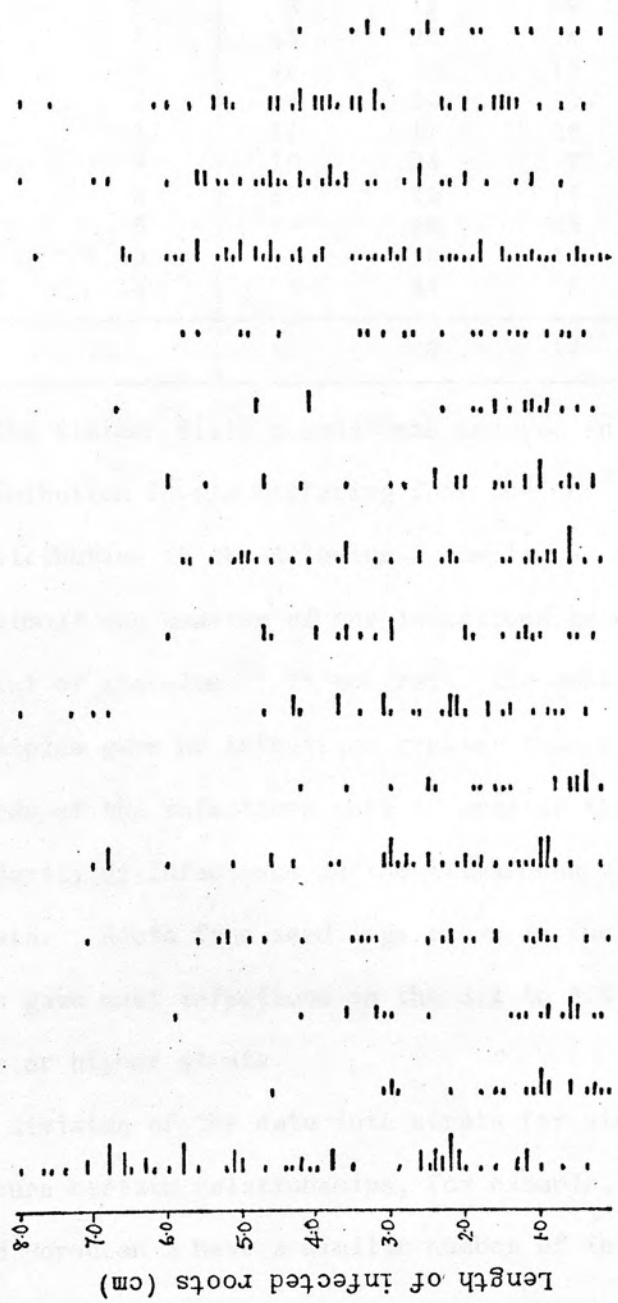


TABLE 9 b: THE EFFECT OF SOIL TEXTURE AND CEREAL HISTORY ON THE DISTRIBUTION OF INFECTIONS IN ASSAYS (PERCENTAGE FREQUENCY OF OBSERVED VALUES)

Soil series	Topsoil texture	Years in cereals	Infection category 0.1–1.0	1.1–2.0	2.1–3.0	3.1–6.0>	6.0 cm
Kilmarnock	CL	1	19	29	26	19	6
		5	23	45	5	23	5
Eckford	S	2	40	36	8	16	0
		2	37	30	4	30	0
		4	14	25	18	39	4
		5	15	26	22	26	11
Alluvium	S	2	3	11	29	33	24
Hobkirk	SL	2	48	24	14	14	0
Belses	CL	2	15	30	15	40	0
Cauldside	C	2	4	20	10	59	6
Darleith	L	3	11	17	28	36	8
Winton	SCL	3	10	24	7	55	3
		8	27	15	15	44	0
Whitsome	C	5	19	25	25	31	0
Horndean	CL	5	13	18	13	48	7
Cessford	CL	5	2	11	18	61	7
Mean			16	22	17	38	7

Of the sixteen field populations assayed in the second group seven had infection levels differing from the chi^2 distribution. The infection distribution in the Alluvium 2 sample was particularly different, with almost one quarter of the infections growing over 6 cm from the point of inoculum. In contrast, the Hobkirk 2 and both Eckford 2 samples gave no infections greater than 6 cm in length and over two-thirds of the infections were no greater than 2 cm. Similarly the majority of infections in the Kilmarnock 5 sample were in the lower strata. Roots from seedlings grown in the Cessford 5 Cauldside 2 soils gave most infections in the 3.1 to 6.0 cm stratum with few in lower or higher strata.

The division of the data into strata for statistical analysis may obscure certain relationships, for example, the assays of Alluvium 2 and Horndean 5 have a similar number of infected roots,

yet figure 39 illustrates that the former has two distinct groups of infection levels falling between 1.0 to 3.0 cm and 5.0 to 7.0 cm whereas the latter has an even population of infections between 0.1 and 3.5 cm and a concentration between 4.0 and 6.0 cm. If another set of samples with an approximately equal number of infections are compared, such as Cauldside 2, Eckford 5, Cessford 5 and Winton 8, contrasting distributions of infection lengths are seen.

Experiment 2: the effect of soil texture and field history

The results of this experiment are shown in Figure 40 and tables 10 a, b and c. The spread of data values is much more concentrated in the lower strata, from 0.1 to 3.0 cm compared with the first experiment. The results of four of the assay samples were not included in the statistical analysis as an inadequate number of roots, less than sixteen, were infected.

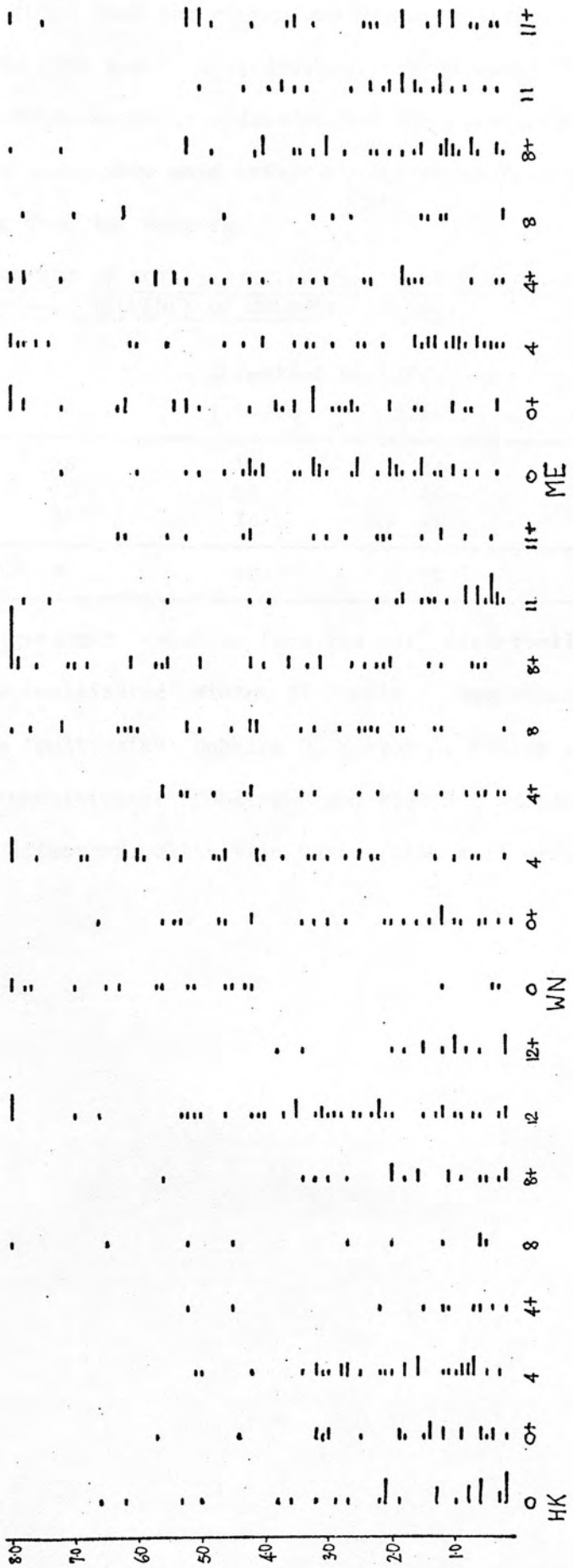
The data was not analysed for differences between cereal histories but the results shown in table 10 a indicate that the distributions of infections in relation to cropping history were very similar.

TABLE 10 a: THE EFFECT OF HISTORY ON THE DISTRIBUTION OF INFECTIONS IN ASSAYS PERCENTAGE FREQUENCY OF OBSERVED VALUES)

Years in cereals	Infection category 0.1-1.0	1.1-2.0	2.1-3.0	> 3.0 cm
0	20	21	15	44
4	21	20	14	45
8	16	19	16	49
11/12	22	27	16	35
Mean	26	20	15	38

Fig 40

The distribution of length of infected roots in the assays of G. graminis populations from three soils of four histories



The data were analysed for overall differences between soils and it was found that the Winton and Macmerry distributions of infection differed from a chi^2 distribution. From table 10 b it can be seen that all three soils gave distinctive distributions: roots from the Macmerry assay were more infected, and those from the Winton less, than from the Hobkirk.

TABLE 10 b: THE EFFECT OF SOIL SERIES ON THE DISTRIBUTION OF INFECTIONS IN ASSAYS (PERCENTAGE FREQUENCY OF OBSERVED VALUES)

	Infection category			
Soil series	0.1-1.0	1.1-2.0	2.1-3.0	> 3.0 cm
Hobkirk	28	17	23	32
Winton	33	35	10	22
Macmerry	20	14	11	56
Mean	26	20	15	38

The greatest deviation from the chi^2 distribution occurred with the 'cultivated' Winton II sample. Deviations also occurred with the 'cultivated' Hobkirk 0, Winton 0, Winton 4 and Macmerry II and 'uncultivated' Hobkirk 0 and Winton 8 samples, showing that the effect of cultivation varied with soil series and history.

TABLE 10 c: THE EFFECT OF CULTIVATION TREATMENT, SOIL SERIES AND HISTORY ON THE DISTRIBUTION OF INFECTIONS IN ASSAYS (PERCENTAGE FREQUENCY OF OBSERVED VALUES)

Treatment	Soil series	Years in cereals	Infection category 0.1-1.0	1.1-2.0	2.1-3.0	> 3.0 cm
Cult	HK	0	42	15	21	21
Cult	HK	4	28	25	22	25
Cult	HK	8	33	11	22	33
Cult	HK	12	15	13	26	46
Non Cult	HK	0	32	36	4	28
Non Cult	HK	4	40	30	10	20
Non Cult	HK	8	32	26	21	21
Non Cult	HK	12	33	47	7	13
Cult	WN	0	13	6	0	81
Cult	WN	4	10	6	16	68
Cult	WN	8	4	15	19	63
Cult	WN	11	50	25	4	21
Non Cult	WN	0	19	27	12	42
Non Cult	WN	4	26	11	11	53
Non Cult	WN	8	9	7	16	67
Non Cult	WN	11	11	17	22	50
Cult	ME	0	7	26	19	48
Cult	ME	4	24	27	11	38
Cult	ME	8	14	29	14	43
Cult	ME	11	11	41	22	26
Non Cult	ME	0	11	11	22	57
Non Cult	ME	4	11	22	7	59
Non Cult	ME	8	21	29	11	39
Non Cult	ME	11	13	33	10	43
Mean			19	21	15	45

Experiment 3: the effect of cereal rotation and nitrogen level.

The results of this experiment are given in figure 41 and table 11.

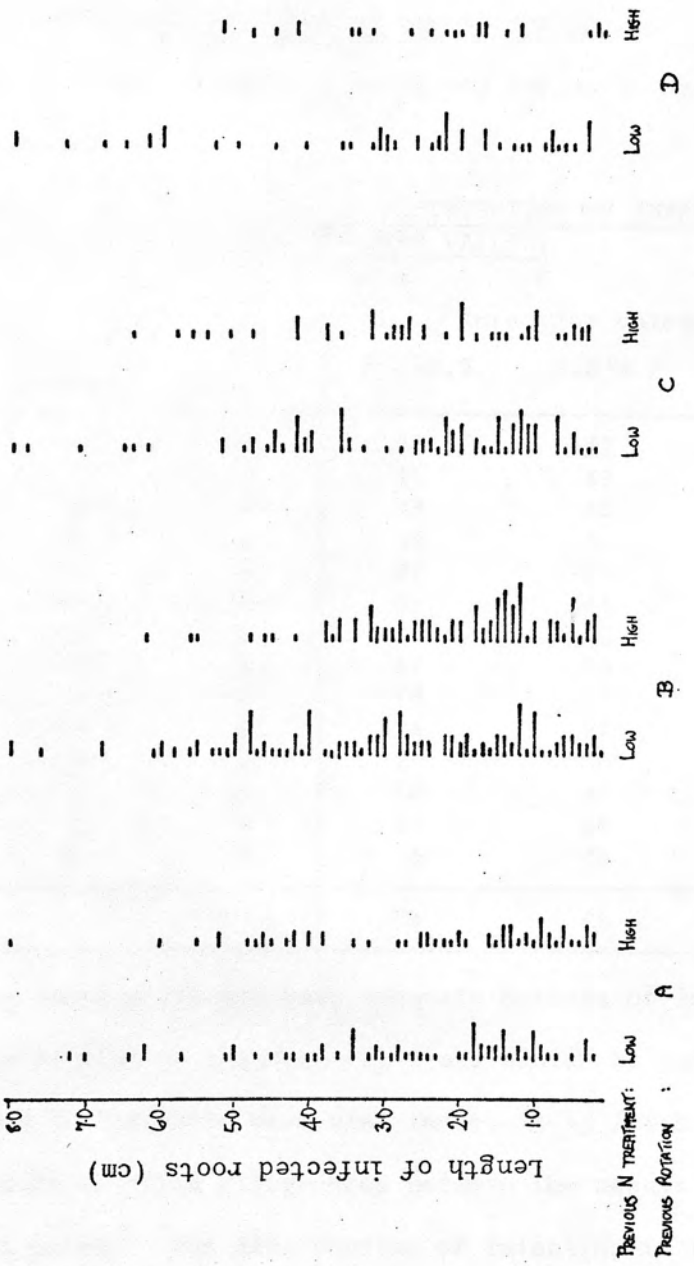
TABLE 11: THE EFFECT OF PREVIOUS CEREAL ROTATION AND NITROGEN LEVEL ON THE DISTRIBUTION OF INFECTIONS IN ASSAYS (PERCENTAGE FREQUENCY OF OBSERVED VALUES)

Rotation	N level	Infection category 0.1-1.5	1.6-3.0	3.1-4.0	> 4.0 cm
A	low	33	28	18	21
A	high	43	27	7	23
B	low	29	25	16	30
B	high	41	36	17	6
C	low	32	31	10	26
C	high	27	39	16	18
D	low	27	35	15	23
D	high	28	36	12	24
		33	31	14	21

Fig 41

The distribution of length of infected roots in the assays of G. graminis populations previously subjected to different cereal rotations and nitrogen treatments

Each millimetre mark represents one infected root.



Analysis of the data indicates that only the infections from the assay of the isolates from plots of rotation B with high nitrogen was significantly different from the chi^2 distribution; all distributions apart from this treatment were very similar to the mean indicating an evenness of isolate behaviour over the remainder of the trial area, 90 x 95 m, and thus no effect of previous nitrogen treatment or host.

Experiment 4: the effect of cereal host.

The results of comparing wheat and barley as assay hosts are shown in table 12.

TABLE 12 : THE EFFECT OF CEREAL HOST ON DISTRIBUTION OF INFECTIONS IN ASSAYS (PERCENTAGE FREQUENCY OF OBSERVED VALUES)

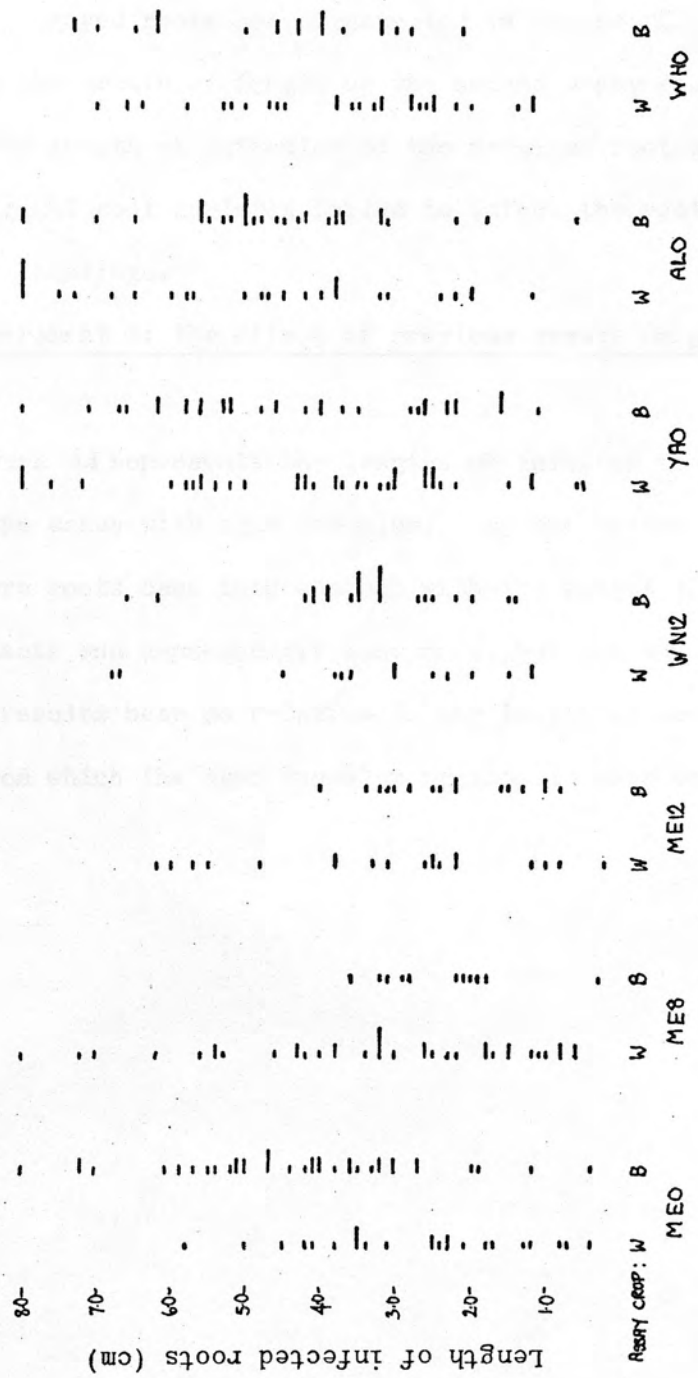
Soil series	Years in cereals	Assay crop	Infection category 0.1-2.5	2.6-4.5	4.5 cm
Macmerry	0	W	46	42	13
	0	B	11	49	41
	8	W	38	41	22
	8	B	55	45	0
	11	W	37	37	26
	11	B	56	44	0
Winton	11	W	36	43	21
	11	B	17	75	8
Yarrow	0	W	18	55	26
	0	B	24	27	48
Alluvium	0	W	22	30	48
	0	B	10	40	50
Whitsome	0	W	18	46	36
	0	B	5	48	48
Mean			24	45	32

Many samples did not have adequate numbers of infections for analysis; one of each of the Macmerry 8 and Winton 11 pairs which are included in the table were also inadequately infected. Analysis of the data revealed differences between the assays of the Macmerry 0 and 11 pairs. The distribution of infection in the

Fig 42

The distribution of length of infected roots in the assays of G. graminis populations in various samples comparing wheat and barley as assay host

Each millimetre mark represents one infected root.



barley assay of the Winton 11 sample was also significantly different from that expected by chance. No statistical differences occur in the other samples, but from figure 42 the distributions of infections in the Macmerry 8 and Yarrow 0 samples appear to differ with assay host.

Experiment 5: the effect of reassaying.

The results for the first repeatability experiment, using previously assayed roots are illustrated in figure 43. In most examples the growth of fungus up the second assay root was not similar to the length of infection of the original root and 10 of the 23 original root isolates failed to infect the roots of the second assay seedlings.

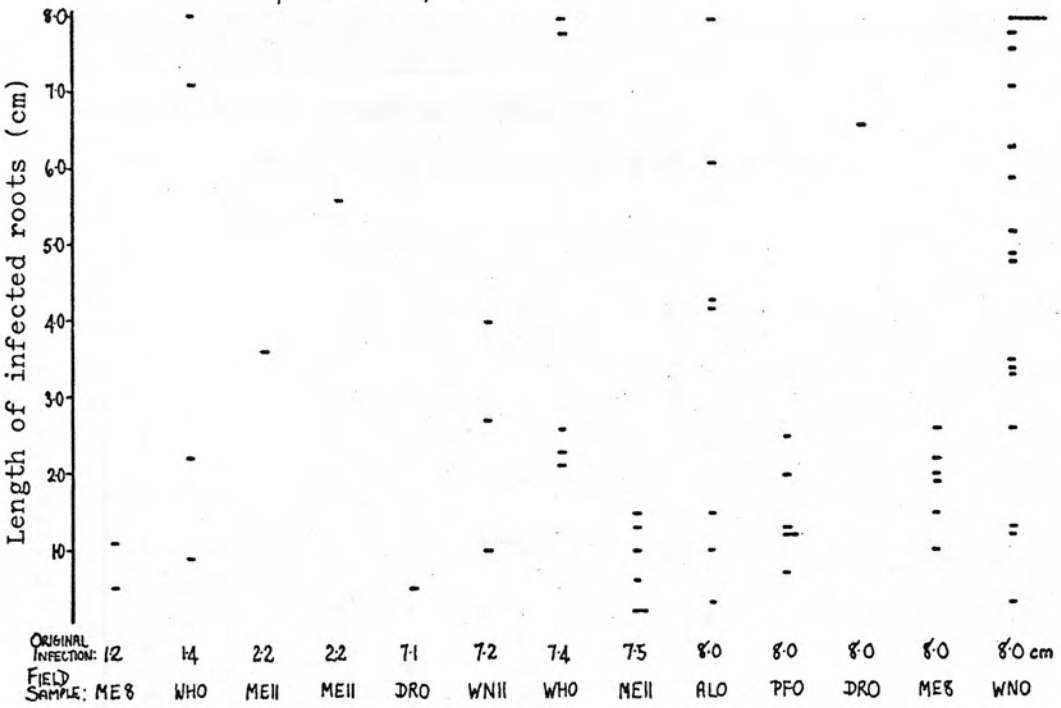
Experiment 6: the effect of previous growth on potato dextrose agar.

Figure 44 represents the lengths of infected root resulting from the assay with agar inoculum. By the nature of the inoculum many more roots came into contact with the fungus than in previous experiments and consequently many more, but not all, were infected. The results bear no relation to the length of originally infected root from which the agar inoculum or inocula were grown.

Fig 43

The distribution of length of infected roots in the reassay of previously infected roots

Each millimetre mark represents one infected root.



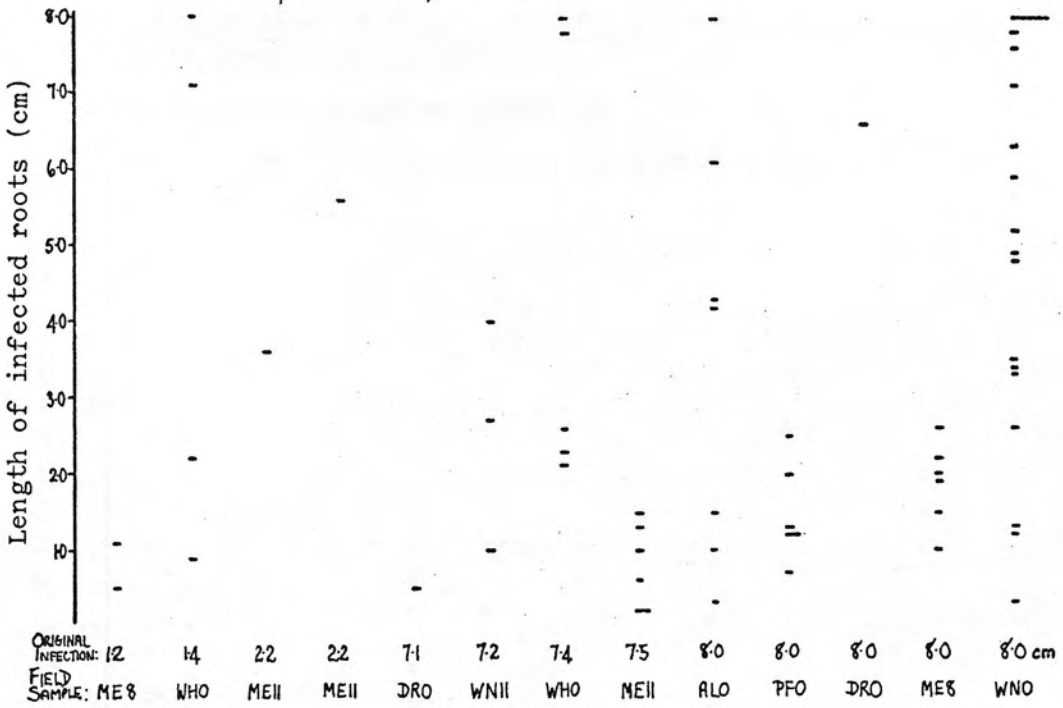
No infections were found in the following reassays:

Field sample	Length of originally infected root (cm)
WN 0	0.3
AL 0	1.2
WN 0	1.3
AL 0	1.4
ME 11	1.5
PF 0	2.0
WN 11	2.1
WH 0	2.1
ME 11	7.5
DR 0	8.0

Fig 43

The distribution of length of infected roots in the reassay of previously infected roots

Each millimetre mark represents one infected root.



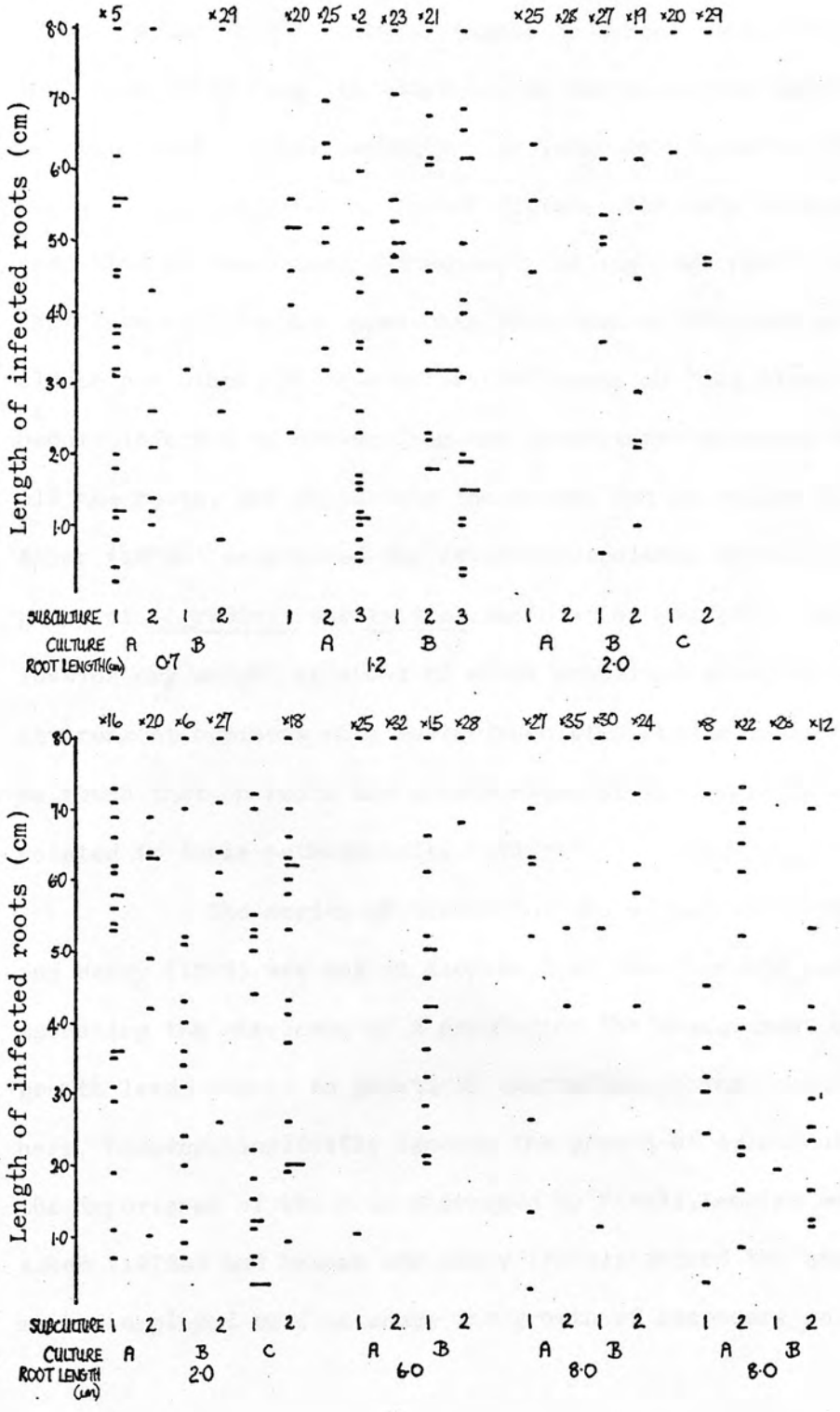
No infections were found in the following reassays:

Field sample	Length of originally infected root (cm)
WN 0	0.3
AL 0	1.2
WN 0	1.3
AL 0	1.4
ME 11	1.5
PF 0	2.0
WN 11	2.1
WH 0	2.1
ME 11	7.5
DR 0	8.0

Fig 44

The distribution of length of infected roots in the reassay of roots using agar inoculum

Each millimetre mark represents one infected root.



Discussion

The validity of this assay technique to test the virulence of a population of isolates of G. graminis as found in agricultural soils rests on the assumption that rate of fungal growth up roots is directly correlated with isolate virulence. The growth of the fungus in the vascular tissue of a root stops the function of that root, including its laterals, at and below the infected area. It also stops further production of laterals: therefore the speed at which the fungus grows up a root dictates the rate of progressive reduction of the functional capacity of its host root. Similarly the closer the fungus approaches the crown of the host plant the closer are other, perhaps uninfected roots on that plant which can become infected by hyphae from the first infected root; in this way all the roots, and ultimately the crown, can be killed by the fungus. Asher (1978b) determined the relative virulence of the members of a group of G. graminis var tritici isolates by recording the percentage loss of dry weight of shoot of wheat seedlings grown in controlled environment cabinets with roots inoculated from colonised agar blocks. He found that on roots the growth rates of the isolates were closely related to their pathogenicity rating.

The merits of the techniques of Asher (1978b) and Deacon and Henry (1978) are not in dispute here, but for the purposes of assessing the virulence of a population the measurement of ectotrophic growth lends itself to practical adaptation. The technique used here, however, implicitly ignores the growth of avirulent isolates, the importance of which is discussed by Tivoli,Lemaire and Jouan (1974), Asher (1978b) and Deacon and Henry (1978); indeed the sterilising method employed to discourage the growth of secondary colonisers would

probably kill many avirulent isolates. Nevertheless, the growth of mildly virulent isolates was measured. It could be argued that if two isolates of different virulence colonise a root, only the isolate growing further up the root will be assessed. However this bias towards the measurement of the most virulent isolates should not occur if the work of Asher (1978b) is correct. He found that preinoculation of a root with a less virulent isolate suppressed growth of more virulent isolates; furthermore, suppressive ability of an isolate was inversely correlated with rate of growth on roots. He thought that rate of ectotrophic growth was possibly a more sensitive measure of isolate virulence than the measurement of percentage loss of shoot dry weight, which failed to differentiate between thirteen hypovirulent isolates he tested. The distribution of lengths of root infections should reflect the relative frequency of more virulent isolates in the sample and the disease potential of the population.

A practical problem associated with this technique was forecasting adequate levels of replication. This was very obvious in experiments 1 and 3 where pooling the data from duplicate pots resulted in no replication. The analysis of data from experiment 3 however, showed that results were similar using inoculum from an area of 90 by 95 m, or 0.855 ha. Consideration of the design of this experiment suggests that probably about 6 pot assays per hectare, comprised of field samples taken in transects, should be adequate for obtaining an estimate of population virulence. If sampling an actively growing crop for inoculum were found to be impossible, assessing stubbles immediately after harvest is acceptable and even using 'bait' seedlings from soil samples, as in experiments 1, 2 and 4 was shown to be feasible from the results of the second experiment.

The use of growth cabinets for this work would greatly improve the uniformity and repeatability of experimental conditions. A more controlled system of watering would be advantageous, particularly when comparing different soil types: bringing the assay pots to a constant weight at regular intervals would be ideal, but time-consuming in a big experiment. Measuring the lengths of infections was found to be no more tedious than many other routine jobs, despite the comments of MacNish, Dodman and Flentje (1973) about its tediousness. Although staining internal hyphae, for example by the Phillips and Hayman (1970) method might be useful for inexperienced workers, the author found it unnecessary. A microscope would be needed to measure non-vascular colonisations.

While recognising the absence of replication in the experiment comparing different soil series, the failure to find a relationship between the infection patterns of soils of a similar texture might suggest that the virulence of the population of isolates in any field is controlled by other factors and may be unique. In some assays in Experiment 2, namely those of Hobkirk 0 and 12, Winton 0 and 11, and probably Winton 4 and Macmerry 11, there were visual differences in results between the previously cultivated and non-cultivated samples. This implies that virulence in these soils, at least at some times under cereal culture, may interact with direct drilling; however the relationship was not consistent in its effect, ie cultivation apparently increased virulence in Hobkirk 12 but decreased it in Winton 11, and more work is needed on this subject. Although no relationship of virulence with cereal history was found this does not mean that virulence may not change with time in any situation.

The result of the experiment comparing the effect of assay crop on the expression of population virulence was inconclusive: however, the data were limited and it is suggested that this subject also merits further investigation. Padwick (1936) and Skou (1968) found no relationship of virulence with the original host, but more recent work by Deacon and Henry (1978), and also by Ao and Griffiths (1976), the latter using Septoria nodorum isolates, indicate differences in expression of virulence and some measure of adaptation to a specific host.

The results of the first reassaying experiment seem inexplicable unless it is assumed that even though the length of an infection may record the growth potential of an isolate, other isolates could have partially colonised or grown ectotrophically on that root-section and have asserted themselves in the reassay; from figure 43 it can be seen that poor repeatability cannot be explained by variation in amount of inoculum alone. In the assays where infection did not reoccur it can be assumed that contact between inoculum and assay root did not take place.

In the 6th experiment previous growth on potato dextrose agar endowed isolates with a markedly increased ability to grow the length of the root; this reaction was unexpected since repeated culturing on agar is generally considered to reduce virulence (eg Chambers, 1970). Without further experimentation it can only be suggested that the sheer volume of inoculum in this experiment over-powered the host's ability to restrict growth, recalling the threshhold elements of Garrett's theory of inoculum potential discussed in Chapter 1.

GENERAL DISCUSSION

This work has been concerned with following the course of take-all disease in a field until its twelfth barley crop. It has also attempted to characterise the development of the disease and assess the virulence of G. graminis populations in soils of contrasting textures and cropping histories.

The observation of take-all disease in the South Road trial revealed several features of interest:

1. The onset of the TAD phenomenon is not fixed in relation to the number of cereal crops grown; in this trial it occurred relatively late.

2. Although the level of the disease varied with nitrogen treatment, cultivation and soil block, the pattern of development was uniform in the trial area.

3. There was no obvious correlation of disease incidence or severity with grain yield.

4. The disease was capable of thriving in conditions of poor crop growth, ie it was most prevalent in the drought year of 1976, and in the plots given no nitrogen fertiliser.

5. Its incidence tended to diminish in conditions conducive to a high population of micro-organisms, ie under minimal cultivation and additions of nitrogen.

6. Severity was not implicated in TAD.

These results suggest that the pattern of development of the disease in this field was dictated by factors within the soil not directly related to its fertility or porosity, as determined by cultivation treatment; although there was no difference in development between the soil blocks it was felt that texture is too important a character to dismiss as a factor affecting development on such limited

evidence. The fact that soil block interacted with cultivation, not nitrogen, in its effect on disease suggests that the disease is more sensitive to physical properties of the soil than to its fertility. This was borne out in the assay of the Macmerry soil from the South Road trial (Chapter 2, Experiment 1): previous nitrogen treatment affected level of infection but not the ultimate development of a decline phase; previous direct drilling, however, changed the pattern of development. The occurrence of TAD does not appear to be affected by disease level as added inoculum failed to alter the infection pattern from that found with natural inoculum in the samples in this experiment. Some innate property of the soil itself and some microbial factor interacting with the host and the fungus may be influencing development, but the interrelationships seem to be complex.

In the second soil assay experiment (Chapter 2, Experiment 2), heating soil at 60^{o}C for 30 minutes before adding inoculum did not change the development of the disease from that of unheated soil with natural or added inoculum. This suggests that any microbial factor present and any natural G. graminis inoculum were not highly heat sensitive.

If, during the assay of a sample, no peak and decline pattern occurred it could be assumed that the soil might already be in a TAD phase. According to Hornby (1978) this is possible even in a field with a short history of cereal-growing, in the sense used in this work, as deliberate attempts to eliminate TAD with 2-year breaks from cereals at Rothamsted failed for no apparent reason. Hornby's finding may partly explain the lack of uniformity of behaviour in assays of samples of the same soil series and history. The impression may be gained that each field is unique in its potential

for take-all development but it would be wrong to ignore the fact that the general TAD pattern was found to occur in the majority of samples and that much of the variation was in the timing and shape of the pattern of disease development.

The assessment of virulence of a field population of G. graminis may indicate the relative likelihood of yield loss at various levels of incidence. Although virulence was not found to be related to cereal history it may be implicated in the long-term development of disease, as Pearson, Hornby and Brown (1973) found some evidence that pathogenicity decreases as take-all develops; particular fields would have to be monitored over a period of years before this information could be ascertained.

The potential for virulence to change may vary with virulence itself, as Chambers and Flentje (1967 a and b) found that this characteristic was correlated with the ability to produce ascospores, and segregration for pathogenicity occurs during sporulation (Hornby, Pearson and Rawlinson, 1973). The potential for virulence to change may also be affected by the soil itself; for example, in a fertile soil greater lysis of fungi occurs than in a less fertile situation (Bumbieris and Lloyd, 1967 b) and so the turnover of isolates may be greater.

In the future simpler methods of assessing virulence may be developed: Asher (1978 b) found some correlation of virulence with hyphal pigmentation, which may develop in response to inhibitory factors (Sivasithamparam and Parker, 1976).

The use of a 28-day seedling assay to predict take-all development and measure population virulence may have a fundamental weakness which is insurmountable: the problem arises because the age

of the host affects the development of G. graminis (Broadfoot, 1933 a). This could be caused by a change in root exudate composition during the growth of the host. Vancura (1964) found biotin amongst the root exudates of wheat and barley seedlings in initial growth; G. graminis has a requirement for biotin (Ward and Henry, 1961). Flück (1955) observed that biotin increases the growth of runner hyphae and this may be the result of a direct effect on wall structure, as Yamaguchi (1974) found that insufficiency of biotin resulted in a marked reduction in thickness of the outermost layer of the cell wall of Candida albicans. Thus the growth of isolates, which would normally have been suppressed because of a relative weakness of cell wall structure predisposing them to lysis (Ballesta and Alexander, 1972), may be abnormally represented by using seedlings in assays.

In the assay of take-all development it is suggested that, in some cases, it would be beneficial to have a greater number of harvests, as eight may be insufficient to reveal a decline in infection level. As this would extend the length of the assay it is therefore pertinent to consider whether a shorter period of growth between harvests would be possible. Wildermuth and Rovira (1977), comparing the suppressiveness of soils, recorded visible differences in hyphal density on seedlings between 7 and 9 days old. Lucas (1963) found that growth of G. graminis var tritici along roots was approximately 1.5 mm/day. On this basis 28 days is theoretically an inadequate period for the fungus to grow from the depth of 8.5 cm to within 0.5 cm of the crown of the host seedling; yet although growth rate on seedlings can obviously be twice that rate, it is felt that in both assay techniques the fungal inoculum should have a chance to develop on the host so that vascular disruption can be measured.

During the period of this work many field samples were examined for take-all disease. Although the cereal-growing areas of East Lothian and the eastern Borders were not surveyed systematically, it was clear that take-all was common in arable land as it was found in the great majority of the fields sampled. This estimate is much in excess of Richardson's figure of 1% incidence of "take-all and other whiteheads" from a survey of Scottish cereal disease (1972) assessed by plating roots and stem-bases of plants on potato-dextrose agar and recording the resulting growth of hyphae after 7 days. Observations of the hyphal growth of G. graminis var tritici from portions of cereal roots on agar revealed that unless the fungus is the sole coloniser of the host material, which is rare, it is usually overtaken within about 5 days by other fungi and bacteria as they are secondary colonisers of the host and grow more vigorously in the saprophytic conditions provided by the agar.

Few of the farmers of the land sampled for this work were aware that their crops were infected and few had any real knowledge of the disease. In this context Hornby (1978) discussed the problems of forecasting the level of take-all disease in any year and found a broad correlation of disease category with weather at Rothamsted Experimental Station. During his discussion he commented:

"it may be that we do not have a forecasting system because the pressures to develop one are not great and the costs prohibitive. If chemical controls were available or a larger proportion of the cereal crop suffered severe take-all, forecasting might be much further advanced." and "Without chemical control, short-term forecasts once the crop is growing in a risk situation would be of limited value because few options are open to the farmer."

The prevalence of take-all implies a considerable potential loss in yield, yet despite its incidence the disease has not often been reported as the major cause of any instance of poor crop health in the region of the East of Scotland College of Agriculture: take-all has

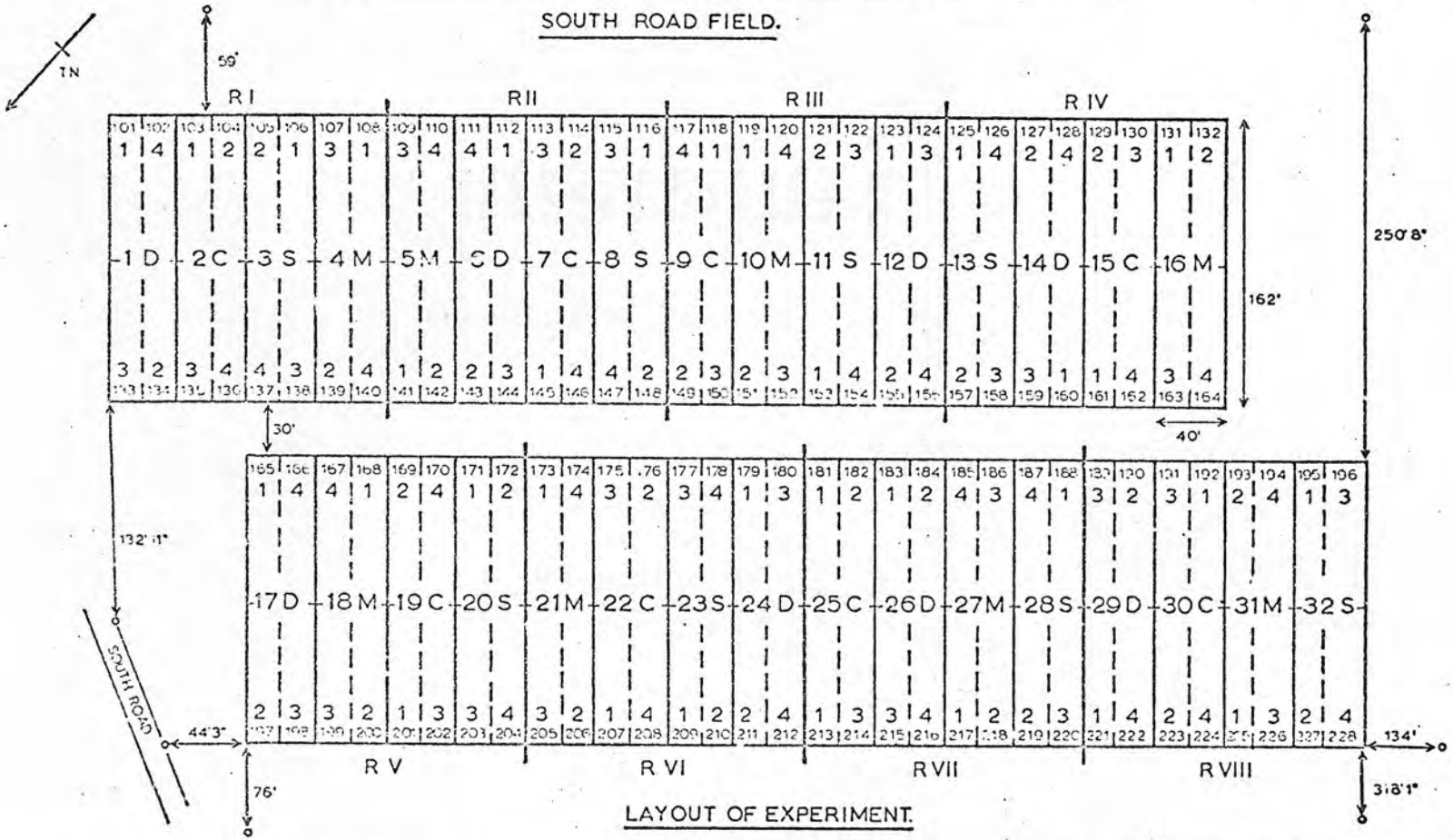
usually been found in conjunction with other problems such as soil drainage, pH and fertility.

Cereal-growing is at a maximum level of intensification in the area under discussion; the only current change in cropping behaviour being an increase in the number of wheat crops grown instead of barley. It is probable that take-all disease is a minor factor contributing to the limitation of possible yields and will never be a major problem in South-East Scotland.

APPENDIX I

CULTIVATION METHODS FOR CONTINUOUS CEREALS EXPERIMENT.

SOUTH ROAD FIELD.



LAYOUT OF EXPERIMENT.

Scale : 1" to 60'

D= DEEP PLOUGHING

S= SHALLOW PLOUGHING

C= CHISEL PLOUGHING

M= MINIMAL CULTIVATION

1 = No N fertiliser

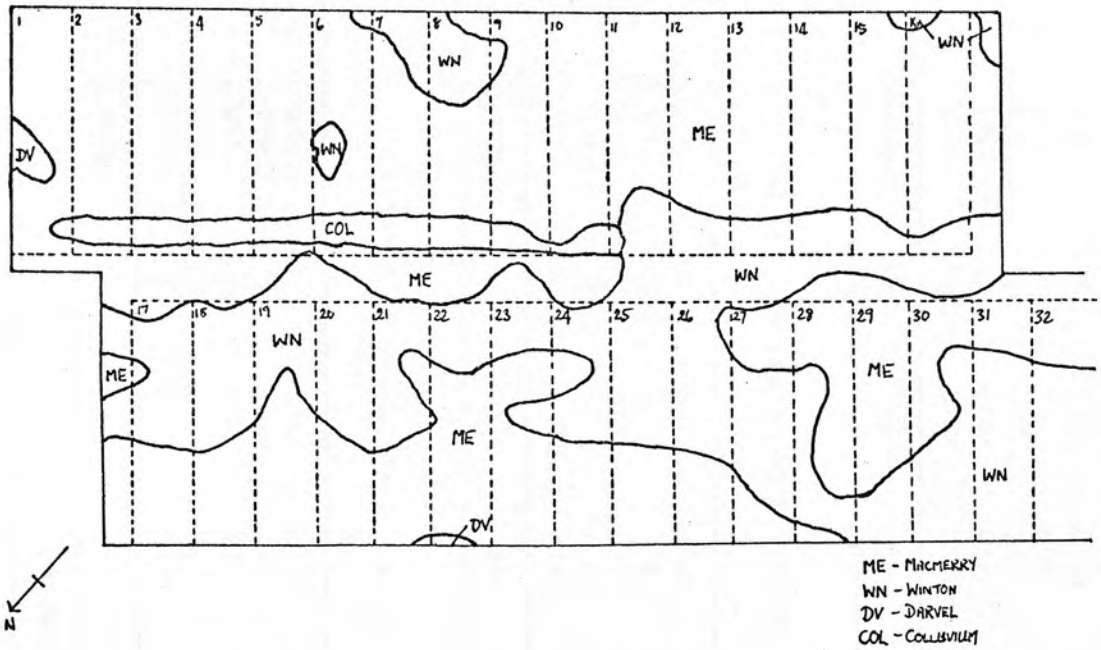
2 = 50 kg N/ha

3 = 100 " " "

4 = 150 " " "

SOIL SERIES DISTRIBUTION IN SOUTH ROAD TRIAL AREA

Surveyed by J M Ragg.



CROPPING HISTORY OF SOUTH ROAD TRIAL AREA

year	crop
1954	oats
1955	swedes
1956	grass
1957	grass
1958	grass
1959	grass
1960	wheat
1961	grass
1962	grass
1963	grass
1964	barley
1965	potatoes
1966 onwards	barley

TAKE-ALL INCIDENCE (% PLANTS INFECTED), CULTIVATION AND NITROGEN TREATMENTS, 1968-1970

1968 I (June/July sample)

	TD	TS	TC	TM	Mean	
NO	4.9	3.5	8.5	4.6	5.4	
N1	2.1	7.5	5.3	3.4	4.6	± 1.10
N2	3.2	3.4	2.1	5.0	3.4	
N3	1.5	2.9	1.5	2.3	2.1	
Mean	2.9	4.3	4.3	3.8	3.9	

± 0.42

Se HI ± 2.00 Se VI ± 2.20 T ns N ns TN ns

II (August sample)

	TD	TS	TC	TM	Mean	
NO	4.7	16.9	21.2	13.2	13.9	
N1	9.5	12.7	8.0	8.2	9.6	± 1.90
N2	8.7	5.4	7.8	5.5	6.8	
N3	3.9	12.1	13.3	3.9	8.3	
Mean	6.7	11.7	12.6	7.7	9.6	

± 1.50

Se HI ± 3.61 Se VI ± 3.80 T * N ns TN ns

1969 I

	TD	TS	TC	TM	Mean	
NO	5.1	13.3	23.2	6.5	12.0	
N1	3.4	5.6	8.1	4.3	5.3	± 1.11
N2	4.2	2.9	4.6	3.1	3.7	
N3	1.6	4.6	3.7	0.8	2.6	
Mean	3.5	6.6	9.9	3.7	5.9	

± 1.06

Se HI ± 2.20 Se VI ± 2.23 T ns N *** TN **

II

	TD	TS	TC	TM	Mean	
NO	19.9	27.7	51.7	11.9	27.8	
N1	8.3	22.4	32.1	15.7	19.6	± 1.94
N2	5.9	9.3	7.0	4.3	6.6	
N3	2.4	8.1	6.6	2.2	4.8	
Mean	9.1	16.9	24.4	8.5	14.7	

± 2.16

Se HI ± 4.00 Se VI ± 3.89 T *** N *** TN ***

1970 I

	TD	TS	TC	TM	Mean	
NO	3.2	20.5	15.0	4.6	10.8	
N1	12.0	9.0	7.8	3.4	8.0	± 1.26
N2	5.5	10.0	9.7	2.8	7.0	
N3	3.4	4.3	7.1	1.5	4.1	
Mean	6.0	10.9	9.9	3.1	7.5	

± 1.00

Se HI ± 2.40 Se VI ± 2.52 T *** N ** TN *

II

	TD	TS	TC	TM	Mean	
NO	8.0	22.9	27.2	7.3	16.3	
N1	9.9	7.5	15.5	2.2	8.7	± 1.76
N2	6.9	5.4	5.0	2.1	4.8	
N3	5.6	8.8	4.1	0.7	4.6	
Mean	7.6	11.0	12.9	3.1	8.6	

± 2.67

Se HI ± 3.80 Se VI ± 3.53 T * N *** TN ns

TAKE-ALL INCIDENCE (% PLANTS INFECTED), CULTIVATION AND NITROGEN TREATMENTS, 1971, 1973 and 1974

1971 I (June/July sample)

	TD	TS	TC	TM	Mean	
N0	7.9	17.9	8.9	5.5	10.1	± 1.31
N1	6.2	8.8	8.6	3.7	6.8	
N2	8.2	8.4	9.7	5.2	7.9	
N3	6.2	10.0	4.9	1.6	5.7	
Mean	7.1	11.3	8.0	4.0	7.6	
		± 1.30				

Se HI ± 2.61 Se VI ± 2.61 T ** Nns TNns

II (August sample)

	TD	TS	TC	TM	Mean	
N0	12.8	25.9	18.4	18.9	18.9	± 2.09
N1	9.1	11.4	26.5	7.5	13.6	
N2	5.5	8.7	6.9	4.1	6.2	
N3	7.9	12.3	5.3	4.3	7.4	
Mean	8.8	14.6	14.3	8.7	11.5	
		± 2.58				

Se HI ± 4.45 Se VI ± 4.19 Tns N *** TN ns

1973 I

	TD	TS	TC	TM	Mean	
N0	14.6	17.3	22.7	17.7	18.1	± 1.66
N1	20.3	15.1	18.7	17.1	17.8	
N2	13.7	11.8	17.7	18.1	15.3	
N3	15.4	15.3	15.8	13.1	14.9	
Mean	16.0	14.9	18.7	16.5	16.5	
		± 1.67				

Se HI ± 3.33 Se VI ± 3.33 T ns N ns TN ns

II

	TD	TS	TC	TM	Mean	
N0	36.7	21.0	41.1	7.6	26.6	± 2.15
N1	23.6	18.6	23.8	18.1	21.0	
N2	15.1	16.5	19.3	10.3	15.3	
N3	15.3	11.0	12.7	10.1	12.3	
Mean	22.7	16.8	24.2	11.5	18.8	
		± 1.51				

Se HI ± 4.03 Se VI ± 4.31 T *** N *** TN *

1974 I

	TD	TS	TC	TM	Mean	
N0	20.8	24.2	19.3	11.5	18.9	± 1.66
N1	16.2	11.4	12.8	5.9	11.6	
N2	12.1	16.2	12.8	11.4	13.1	
N3	13.8	16.5	8.3	22.0	15.2	
Mean	15.7	17.1	13.3	12.7	14.7	
		± 2.05				

Se HI ± 3.54 Se VI ± 3.32 T ns N * TN **

II

	TD	TS	TC	TM	Mean	
N0	48.6	53.3	57.1	25.3	46.0	± 2.31
N1	36.0	40.3	40.5	20.7	34.3	
N2	31.3	35.3	29.8	18.9	28.8	
N3	37.2	30.9	33.2	29.2	32.6	
Mean	38.3	39.9	40.2	23.5	35.4	
		± 2.07				

Se HI ± 4.50 Se VI ± 4.61 T *** N *** TN ns

TAKE-ALL INCIDENCE (% PLANTS INFECTED), CULTIVATION AND NITROGEN TREATMENTS, 1975-1977

1975 I (June/July sample)

	TD	TS	TC	TM	Mean	
N0	60.7	47.4	44.1	38.8	47.7	
N1	44.3	33.8	31.7	44.0	38.4	± 2.74
N2	40.0	35.0	33.5	56.7	41.3	
N3	43.2	31.8	29.4	54.0	39.6	
Mean	47.0	37.0	34.7	48.4	41.8	
		± 2.66				

Se HI ± 5.44 Se VI ± 5.48 T ** N ns TN **

II (August sample)

	TD	TS	TC	TM	Mean	
N0	86.2	63.8	69.0	37.6	64.1	
N1	48.6	52.1	57.3	33.1	47.1	± 3.40
N2	43.8	33.3	37.4	25.8	35.0	
N3	46.5	27.7	25.0	30.7	32.4	
Mean	56.3	44.2	47.2	31.8	44.9	
		± 3.69				

Se HI ± 6.95 Se VI ± 6.80 T ** N *** TN ns

1976 I

	TD	TS	TC	TM	Mean	
N0	47.2	47.1	42.9	44.3	45.4	
N1	40.8	48.8	31.9	37.7	39.8	± 3.58
N2	37.8	35.1	35.1	44.7	38.1	
N3	45.3	42.5	35.8	39.4	40.8	
Mean	42.8	43.4	36.4	41.5	41.0	
		± 2.59				

Se HI ± 6.72 Se VI ± 7.16 T ns N ns TN ns

II

	TD	TS	TC	TM	Mean	
N0	82.3	86.1	90.1	79.5	84.5	
N1	86.9	85.5	88.9	74.9	84.1	± 2.11
N2	70.4	84.7	77.6	78.9	77.8	
N3	78.2	75.2	80.5	77.7	77.9	
Mean	79.5	82.9	84.3	77.7	81.0	
		± 2.44				

Se HI ± 4.39 Se VI ± 4.22 T ns N * TN ns

1977 I

	TD	TS	TC	TM	Mean	
N0	50.7	32.9	42.4	15.9	35.5	
N1	22.9	26.3	39.8	18.9	27.0	± 2.40
N2	18.2	17.4	15.9	20.1	17.9	
N3	23.7	13.1	14.3	18.7	17.5	
Mean	28.9	22.4	28.1	18.4	24.5	
		± 2.77				

Se HI ± 4.99 Se VI ± 4.79 T * N *** TN **

II

	TD	TS	TC	TM	Mean	
N0	83.4	63.2	76.5	44.9	67.0	
N1	47.5	44.8	52.1	42.3	46.7	± 2.47
N2	31.4	27.0	37.5	38.4	33.6	
N3	34.6	17.1	35.7	34.2	30.4	
Mean	49.2	38.0	50.5	39.9	44.4	
		± 1.62				

Se HI ± 4.57 Se VI ± 4.94 T *** N *** TN **

TAKE-ALL INCIDENCE, % PLANTS INFECTED, SOIL BLOCK v NITROGEN AND CULTIVATION TREATMENTS, 1968-1970

1968 I (June/July sample)	no infected roots/plant		II (August sample)		
	S1	S2		S1	S2
N0	3.1	7.7	N0	6.3	21.7
N1	3.0	6.2	N1	2.3	17.0
N2	3.4	3.5	N2	2.8	10.9
N3	1.9	2.2	N3	3.3	13.2
TD	2.5	3.3	TD	1.1	12.3
TS	2.0	6.6	TS	5.4	18.1
TC	3.2	5.5	TC	4.2	21.0
TM	3.6	4.0	TM	4.0	11.4
Mean	2.9	4.9	Mean	3.7	15.7
	± 0.42			± 1.06	
N: Se H ± 1.41 Se V ± 1.56			Se H ± 2.55 Se V ± 2.67		
T: Se ± 0.84			Se ± 2.11		
S ** SN ns ST ns			S *** SN ns ST ns		

1969 I			II		
	S1	S2		S1	S2
N0	10.1	14.0	N0	19.1	36.5
N1	3.3	7.4	N1	12.5	26.8
N2	2.9	4.6	N2	3.5	9.8
N3	3.1	2.2	N3	4.0	5.7
TD	3.2	3.9	TD	5.0	13.3
TS	5.0	8.2	TS	10.3	23.5
TC	8.3	11.5	TC	20.1	28.7
TM	2.9	4.5	TM	3.8	13.3
Mean	4.8	7.0	Mean	9.8	19.7
	± 0.75			± 1.53	
N: Se H ± 1.56 Se V ± 1.58			Se H ± 2.83 Se V ± 2.75		
T: Se ± 1.50			Se ± 3.05		
S ns SN ns ST ns			S *** SN ns ST ns		

1970 I			II		
	S1	S2		S1	S2
N0	14.8	6.9	N0	22.5	10.2
N1	8.7	7.4	N1	10.7	6.9
N2	8.6	5.4	N2	5.9	3.8
N3	5.2	3.0	N3	5.8	3.5
TD	6.1	5.9	TD	8.8	6.4
TS	14.1	77.8	TS	14.7	7.4
TC	14.5	5.3	TC	18.1	7.7
TM	2.6	3.5	TM	3.3	2.9
Mean	9.3	5.7	Mean	11.2	6.1
	± 0.71			± 1.60	
N: Se H ± 1.70 Se V ± 1.78			Se H ± 2.69 Se V ± 2.49		
T: Se ± 1.41			Se ± 3.20		
S * SN ns ST *			S ns SN ns ST ns		

TAKE-ALL INCIDENCE, % PLANTS INFECTED, SOIL BLOCK v NITROGEN AND CULTIVATION TREATMENTS, 1971, 1973 AND 1974

1971 I (June/July sample)

	S1	S2
N0	15.8	4.3
N1	10.4	3.2
N2	11.6	4.1
N3	8.5	2.8
TD	11.1	3.2
TS	16.5	6.1
TC	12.7	3.3
TM	6.0	2.0
Mean	11.6	3.6

± 0.92

N: Se H ± 1.85 Se V ± 1.85
T: Se ± 1.83
S *** SN ns ST ns

II (August sample)

	S1	S2
N0	21.3	16.7
N1	13.7	13.5
N2	8.0	4.5
N3	8.8	6.1
TD	12.3	5.4
TS	18.4	10.7
TC	14.6	13.9
TM	6.6	10.8
Mean	13.0	10.2

± 1.83

Se H ± 3.15 Se V ± 2.96
Se ± 3.65
S ns SN ns ST ns

1973 I

	S1	S2
N0	21.2	15.0
N1	20.2	15.4
N2	19.0	12.0
N3	18.4	11.3
TD	21.0	11.0
TS	20.0	9.9
TC	21.3	16.2
TM	16.3	16.7
Mean	19.6	13.4

± 1.45

N: Se H ± 2.37 Se V ± 2.50
T: Se ± 2.51
S ** SN ns TS ns

II

	S1	S2
N0	28.3	25.0
N1	18.4	23.7
N2	14.1	16.5
N3	11.6	13.0
TD	26.5	18.9
TS	21.0	12.5
TC	19.2	29.3
TM	5.7	17.3
Mean	18.1	19.5

± 1.07

Se H ± 2.85 Se V ± 3.05
Se ± 2.13
S ns SN ns ST ***

1974 I

	S1	S2
N0	22.9	15.0
N1	13.0	10.1
N2	14.8	11.4
N3	17.5	12.9
TD	19.6	11.8
TS	21.3	12.9
TC	15.3	11.3
TM	11.9	13.5
Mean	17.0	12.4

± 1.45

N: Se H ± 2.50 Se V ± 2.35
T: Se ± 2.91
S ns SN ns ST *

II

	S1	S2
N0	52.9	39.3
N1	41.9	26.9
N2	35.3	22.3
N3	38.4	26.9
TD	48.8	27.8
TS	50.0	29.9
TC	46.4	34.0
TM	23.3	23.7
Mean	42.1	28.8

± 1.46

Se H ± 3.18 Se V ± 3.26
Se ± 2.93
S *** SN ns ST *

TAKE-ALL INCIDENCE, % PLANTS INFECTED, SOIL BLOCK v NITROGEN AND CULTIVATION TREATMENTS, 1975-1977

1975	I (June/July sample)			II (August sample)	
	S1	S2		S1	S2
N0	49.8	45.6	N0	68.5	59.8
N1	36.9	40.0	N1	50.6	44.9
N2	43.4	39.3	N2	43.4	26.8
N3	40.5	38.7	N3	39.9	24.9
TD	53.0	41.1	TD	63.5	49.0
TS	40.5	33.5	TS	43.3	45.1
TC	36.7	32.6	TC	56.7	37.7
TM	40.4	56.3	TM	39.0	24.9
Mean	42.7	40.9	Mean	50.6	39.1
	± 1.88			± 2.61	
N: Se H ± 3.85 Se V ± 3.88			Se H ± 4.92 Se V ± 4.81		
T: Se ± 3.77			Se ± 5.22		
S ns SN ns ST **			S ** SN ns ST ns		

1976	I			II	
	S1	S2		S1	S2
N0	44.6	47.3	N0	87.9	83.2
N1	38.9	40.1	N1	82.9	84.2
N2	30.9	45.0	N2	74.7	80.4
N3	36.7	44.5	N3	73.9	81.3
TD	44.9	40.6	TD	79.1	79.5
TS	39.3	47.4	TS	83.1	82.6
TC	30.4	42.4	TC	80.8	87.7
TM	36.5	46.5	TM	76.5	79.0
Mean	37.8	44.2	Mean	79.9	82.3
	± 1.83			± 1.72	
N: Se H ± 4.75 Se V ± 5.06			Se H ± 3.11 Se V ± 2.99		
T: Se ± 3.67			Se ± 3.45		
S * SN ns ST ns			S ns SN ns ST ns		

1977	I			II	
	S1	S2		S1	S2
N0	35.2	35.8	N0	65.1	68.9
N1	26.0	27.9	N1	39.3	54.0
N2	16.3	19.5	N2	31.2	36.0
N3	11.3	23.7	N3	28.6	32.2
TD	28.2	29.5	TD	49.3	49.2
TS	23.0	21.9	TS	33.2	42.9
TC	24.7	31.5	TC	49.9	51.1
TM	12.8	24.0	TM	31.8	48.1
Mean	22.2	26.7	Mean	41.0	47.8
	± 1.96			± 1.14	
N: Se H ± 3.53 Se V ± 3.39			Se H ± 3.23 Se V ± 3.49		
T: Se ± 3.91			Se ± 2.28		
S ns SN ns ST ns			S ** SN ns ST **		

TAKE-ALL SEVERITY, CULTIVATION AND NITROGEN TREATMENTS, 1968-1969

1968 I (June/July sample) no infected roots/plant II (August sample) Index - see p 47

	TD	TS	TC	TM	Mean	
N0	1.00	0.71	1.20	0.82	0.93	
N1	0.56	0.80	1.02	0.95	0.83	± 0.141
N2	0.91	0.86	0.84	1.01	0.91	
N3	0.66	1.35	0.37	1.12	0.88	
Mean	0.78	0.93	0.86	0.98	0.89	
		± 0.105				

Se HI ± 0.266 Se VI ± 0.282 T ns N ns TN ns

	TD	TS	TC	TM	Mean	
N0	0.51	1.17	1.16	0.76	0.90	
N1	0.50	0.64	0.66	0.50	0.57	± 0.066
N2	0.71	0.79	0.87	1.06	0.86	
N3	0.65	1.01	0.90	0.87	0.86	
Mean	0.59	0.90	0.90	0.80	0.80	
		± 0.090				

Se HI ± 0.146 Se VI ± 0.132 T ns N ** TN ns

1969 I II

	TD	TS	TC	TM	Mean	
N0	0.59	1.34	1.49	0.90	1.08	
N1	0.82	1.42	1.39	0.62	1.07	± 0.126
N2	1.36	0.80	0.99	1.10	1.06	
N3	0.69	1.07	0.65	0.37	0.70	
Mean	0.87	1.16	1.13	0.75	0.98	
		± 0.155				

Se HI ± 0.268 Se VI ± 0.252 T ns N ns TN ns

	TD	TS	TC	TM	Mean	
N0	1.62	1.62	2.10	1.41	1.69	
N1	1.07	1.54	1.47	1.57	1.41	± 0.130
N2	0.66	1.10	1.09	0.59	0.86	
N3	0.70	0.80	0.70	0.37	0.64	
Mean	1.02	1.27	1.34	0.99	1.15	
		± 0.162				

Se HI ± 0.277 Se VI ± 0.260 T ns N *** TN ns

TAKE-ALL SEVERITY, CULTIVATION AND NITROGEN TREATMENTS, 1970 and 1973

1970 I (June/July sample)

	TD	TS	TC	TM	Mean	
N0	1.05	1.54	1.49	0.74	1.20	± 0.122
N1	1.42	1.26	1.64	0.94	1.32	
N2	1.02	1.56	1.10	0.62	1.08	
N3	0.92	1.27	1.15	0.72	1.02	
Mean	1.10	1.41	1.34	0.76	1.15	

± 0.087

Se HI ± 0.173 Se VI ± 0.173 T * N ns TN ns

II (August sample)

	TD	TS	TC	TM	Mean	
N0	1.20	1.16	1.11	1.29	1.19	± 0.073
N1	1.00	1.01	1.11	0.50	0.91	
N2	1.01	0.79	0.65	0.75	0.80	
N3	0.64	0.67	0.52	0.25	0.52	
Mean	0.96	0.91	0.85	0.70	0.85	

± 0.073

Se HI ± 0.145 Se VI ± 0.145 T ns N *** TN ns

1973

TD	TS	TC	TM	Mean
No Assessment made				

II

	TD	TS	TC	TM	Mean	
N0	1.63	1.20	1.85	1.28	1.49	± 0.11
N1	1.50	1.83	1.42	1.41	1.54	
N2	1.95	1.40	1.68	1.51	1.63	
N3	1.18	1.41	1.12	1.38	1.27	
Mean	1.56	1.46	1.52	1.40	1.49	

± 0.09

Se HI ± 0.22 Se VI ± 0.23 T ns N ns TN ns

TAKE-ALL SEVERITY, CULTIVATION AND NITROGEN TREATMENTS, 1975-1977

1975 I (June/July sample)

	TD	TS	TC	TM	Mean	
N0	2.70	3.07	2.84	2.31	2.73	± 0.11
N1	2.13	2.06	1.92	2.13	2.06	
N2	2.19	2.19	1.74	2.25	2.09	
N3	2.31	2.20	2.06	1.99	2.14	
Mean	2.33	2.38	2.14	2.17	2.26	
			± 0.15			

Se HI ± 0.24 Se VI ± 0.22 T ns N *** TN ns

II (August sample)

	TD	TS	TC	TM	Mean	
N0	1.38	1.31	1.27	1.24	1.30	± 0.04
N1	1.11	1.20	1.30	1.23	1.21	
N2	1.09	1.10	1.11	1.18	1.12	
N3	1.13	1.07	0.92	1.26	1.08	
Mean	1.18	1.17	1.15	1.23	1.18	
			± 0.05			

Se HI ± 0.09 Se VI ± 0.09 T ns N ** TN ns

1976 I

	TD	TS	TC	TM	Mean	
N0	2.40	2.17	2.76	2.34	2.42	± 0.09
N1	2.23	2.66	2.69	2.40	2.50	
N2	2.19	2.44	2.11	2.45	2.30	
N3	2.59	2.55	2.42	2.58	2.53	
Mean	2.35	2.45	2.49	2.44	2.44	
			± 0.05			

Se HI ± 0.18 Se VI ± 0.19 T ns N ns TN ns

II

	TD	TS	TC	TM	Mean	
N0	1.69	1.69	1.90	1.68	1.74	± 0.04
N1	1.65	1.67	1.81	1.70	1.71	
N2	1.61	1.67	1.70	1.84	1.70	
N3	1.64	1.54	1.66	1.64	1.62	
Mean	1.65	1.64	1.77	1.71	1.69	
			± 0.04			

Se HI ± 0.08 Se VI ± 0.08 T ns N ns TN ns

1977 I

	TD	TS	TC	TM	Mean	
N0	2.56	1.93	2.58	2.15	2.31	± 0.14
N1	1.99	1.95	2.18	2.00	2.03	
N2	1.90	2.39	1.90	1.53	1.93	
N3	2.47	1.91	1.69	2.60	2.16	
Mean	2.23	2.04	2.09	2.07	2.11	
			± 0.13			

Se HI ± 0.27 Se VI ± 0.28 T ns N ns TN ns

II

	TD	TS	TC	TM	Mean	
N0	1.70	1.52	1.76	1.86	1.71	± 0.07
N1	1.35	1.46	1.48	1.69	1.50	
N2	1.73	1.41	1.57	1.78	1.62	
N3	1.42	1.77	1.65	2.11	1.74	
Mean	1.55	1.54	1.62	1.86	1.64	
			± 0.05			

Se HI ± 0.13 Se VI ± 0.13 T ** N ns TN ns

TAKE-ALL SEVERITY, SOIL BLOCK v NITROGEN AND CULTIVATION TREATMENTS, 1968 AND 1969

1968 I (June/July sample) no.infected roots/plant

	S1	S2
N0	0.76	1.11
N1	0.84	0.82
N2	0.87	0.94
N3	0.71	1.05
TD	0.75	0.82
TS	0.64	1.22
TC	0.91	0.81
TM	0.89	1.06
Mean	0.80	0.98

± 0.074

N: Se H ± 0.188 Se V ± 0.199
T: Se ± 0.149
S ns SN ns ST ns

1968 II (August sample) Index - see p 47.

	S1	S2
N0	0.76	1.04
N1	0.31	0.84
N2	0.64	1.07
N3	0.69	1.03
TD	0.27	0.91
TS	0.79	1.01
TC	0.77	1.02
TM	0.56	1.04
Mean	0.60	1.00

± 0.064

Se H ± 0.103 Se V ± 0.094
Se ± 0.127
S *** SN ns ST ns

1969 I

	S1	S2
N0	1.07	1.08
N1	1.11	1.02
N2	0.95	1.17
N3	0.67	0.72
TD	0.86	0.87
TS	0.90	1.42
TC	1.24	1.01
TM	0.81	0.69
Mean	0.95	1.00

± 0.110

N: Se H ± 0.189 Se V ± 0.178
T: Se ± 0.219
S ns SN ns St ns

1969 II

	S1	S2
N0	1.66	1.72
N1	1.16	1.67
N2	0.66	1.06
N3	0.47	0.81
TD	0.73	1.30
TS	1.19	1.34
TC	1.16	1.52
TM	0.87	1.10
Mean	0.99	1.31

± 0.114

Se H ± 0.196 Se V ± 0.184
Se ± 0.229
S ns SN ns ST ns

TAKE-ALL SEVERITY, SOIL BLOCK v NITROGEN AND CULTIVATION TREATMENTS, 1970 AND 1973

1970	I (June/July sample)			II (August sample)	
	S1	S2		S1	S2
N0	1.34	1.07	N0	1.38	1.00
N1	1.29	1.34	N1	0.85	0.96
N2	1.19	0.96	N2	0.77	0.83
N3	1.11	0.92	N3	0.46	0.58
TD	1.19	1.02	TD	0.98	0.94
TS	1.50	1.32	TS	0.97	0.84
TC	1.57	1.12	TC	0.89	0.81
TM	0.67	0.84	TM	0.62	0.77
Mean	1.23	1.07	Mean	0.87	0.84
	± 0.087			± 0.051	
N: Se H ± 0.173 Se V ± 0.173			Se H ± 0.103 Se V ± 0.103		
T: Se ± 0.173			Se ± 0.103		
S ns SN ns ST ns			S ns SN * ST ns		

1973			II		
	S1	S2		S1	S2
No Assessment made			N0	1.32	1.67
			N1	1.47	1.61
			N2	1.37	1.90
			N3	1.23	1.32
			TD	1.56	1.57
			TS	1.40	1.52
			TC	1.33	1.71
			TM	1.09	1.70
			Mean	1.34	1.63
				± 0.066	
			N: Se H ± 0.156 Se V ± 0.163		
			T: Se ± 0.133		
			S ** SN ns ST ns		

TAKE-ALL SEVERITY, SOIL BLOCK v NITROGEN AND CULTIVATION TREATMENTS, 1975-1977

1975 I (June/July sample)

	S1	S2
NO	2.95	2.51
N1	2.09	2.03
N2	2.06	2.13
N3	2.31	1.98
TD	2.54	2.12
TS	2.59	2.16
TC	2.20	2.08
TM	2.07	2.28
Mean	2.35	2.16

± 0.107

N: Se H ± 0.170 Se V ± 0.152
T: Se ± 0.215
S ns SN ns ST ns

II (August sample)

	S1	S2
NO	1.31	1.30
N1	1.28	1.14
N2	1.13	1.11
N3	1.18	1.01
TD	1.23	1.12
TS	1.15	1.19
TC	1.28	1.03
TM	1.24	1.22
Mean	1.22	1.14

± 0.037

Se H ± 0.065 Se V ± 0.062
Se ± 0.074
S ns SN ns ST ns

1976 I

	S1	S2
NO	2.42	2.39
N1	2.39	2.60
N2	2.13	2.50
N3	2.35	2.73
TD	2.24	2.46
TS	2.33	2.58
TC	2.41	2.58
TM	2.33	2.55
Mean	2.33	2.55

± 0.055

N: Se H ± 0.126 Se V ± 0.131
T: Se ± 0.111
S* SN ns ST ns

II

	S1	S2
NO	1.77	1.72
N1	1.63	1.78
N2	1.60	1.79
N3	1.51	1.74
TD	1.63	1.67
TS	1.55	1.73
TC	1.69	1.85
TM	1.64	1.79
Mean	1.63	1.76

± 0.028

Se H ± 0.054 Se V ± 0.053
Se ± 0.055
S ** SN ns ST ns

1977 I

	S1	S2
NO	2.44	2.17
N1	1.75	2.31
N2	1.74	2.12
N3	1.68	2.65
TD	2.23	2.23
TS	1.85	2.24
TC	1.71	2.47
TM	1.83	2.31
Mean	1.90	2.31

± 0.092

N: Se H ± 0.193 Se V ± 0.195
T: Se ± 1.84
S ** SN * ST ns

II

	S1	S2
NO	1.72	1.70
N1	1.40	1.59
N2	1.51	1.73
N3	1.68	1.80
TD	1.55	1.55
TS	1.39	1.70
TC	1.62	1.62
TM	1.76	1.96
Mean	1.58	1.71

± 0.038

Se H ± 0.090 Se V ± 0.094
Se ± 0.076
S * SN ns ST ns

THE YIELD OF BARLEY (t/ha) AT 85% MOISTURE FROM THE SOUTH ROAD TRIAL, 1966 to 1977

Year		1966	1967	1968	1969	1970	1971	1972	1973	1974	1975	1976	1977	mean of 1968 to 1977
Nitrogen level	N0			1.51	2.43	2.46	2.16	2.23	2.54	2.62	1.59	1.64	1.28	2.05
	N1			2.79	3.82	3.61	3.05	3.83	3.53	4.37	3.75	2.97	3.08	3.48
	N2			3.43	4.54	4.17	2.95	5.03	3.85	5.27	4.53	3.62	4.72	4.21
	N3			3.53	5.02	4.58	2.88	5.36	3.93	5.77	4.88	3.57	5.03	4.45
Cultivation	Deep			2.90	4.04	3.70	2.83	4.31	3.72	4.76	3.71	2.93	3.73	3.66
	Shallow			2.92	4.14	3.99	2.90	4.57	3.68	4.60	3.77	3.00	3.60	3.72
	Chisel			2.84	3.93	3.64	2.78	4.17	3.53	4.26	3.51	2.86	3.29	3.48
	Minimal			2.61	3.70	3.48	2.55	3.42	2.91	4.39	3.75	3.00	3.51	3.33
Soil Block	1	4.03	6.05	2.99	4.04	3.57	2.75	4.50	3.54	4.57	3.86	3.03	3.57	3.64
	2	3.91	5.41	2.65	3.87	3.84	2.77	3.73	3.39	4.44	3.52	2.87	3.49	3.46
SE (difference) soils		± 0.03	0.05	0.03	0.06	0.05	0.03	0.05	0.05	0.06	0.05	0.04	0.09	
Mean		3.97	5.73	2.82	3.95	3.70	2.76	4.12	3.47	4.51	3.69	2.95	3.53	3.55

APPENDIX II

EXPERIMENT 1: TAKE-ALL INFECTION (cm per root) from HARVESTS 4 to 10; PRIOR CULTIVATION AND NITROGEN TREATMENTS

Cultivation	harvest 4	5	6	7	8	9	10	Mean
Deep	5.27	4.25	2.92	3.46	3.90	2.37	1.28	3.35
Chisel	5.09	5.43	2.81	3.87	3.76	2.49	1.69	3.59
Shallow	3.57	4.77	3.42	3.59	4.52	2.85	2.49	3.60
Minimal	2.32	2.72	1.77	1.95	2.50	1.07	0.83	1.88
Nitrogen								
0	4.66	5.49	3.37	3.80	3.57	2.08	1.53	3.50
+N	3.47	3.09	2.09	2.63	3.77	2.31	1.61	2.71
Mean	4.06	4.29	2.73	3.22	3.67	2.20	1.57	3.11

SE (difference) nitrogen level ± 0.327 *
cultivations ± 0.314 **
harvest ± 0.386 ***

nitrogen level v harvest: nitrogen level ± 0.546
harvest ± 0.602 **
cultivation v harvest: cultivation ± 0.772 ns
harvest ± 0.781

EXPERIMENT 1: TAKE-ALL INFECTION (cm per root) from HARVESTS 4 to 10; PRIOR CULTIVATION AND NITROGEN TREATMENTS

Cultivation	Nitrogen	harvest 4	5	6	7	8	9	10	Mean
Deep	0	5.91	5.23	3.71	4.02	3.66	2.25	1.44	3.75
	+N	4.62	3.27	2.12	2.89	4.14	2.48	1.11	2.95
Chisel	0	4.59	5.17	2.77	3.68	3.42	1.59	1.26	3.21
	+N	5.59	5.68	2.85	4.06	4.10	3.40	2.12	3.97
Shallow	0	4.54	6.69	3.82	4.20	3.38	2.85	2.07	3.94
	+N	2.59	2.86	3.02	2.98	5.66	2.84	2.91	3.26
Minimal	0	3.57	4.87	3.17	3.30	3.83	1.64	1.36	3.11
	+N	1.07	0.57	0.38	0.60	1.17	0.51	0.30	0.66
Mean		4.06	4.29	2.73	3.22	3.67	2.20	1.57	3.11

SE (difference) means:

harvest		± 0.386 ***
cultivations v nitrogen level:	cultivations	± 0.654
	nitrogen level	± 0.559 *
cultivation v nitrogen level v harvest		± 1.155 ns

EXPERIMENT 2: TAKE-ALL INFECTION (transformed data), HARVESTS 1 to 8 WITH EXPERIMENTAL TREATMENTS ON DIFFERENT SOIL TEXTURES AND CEREAL HISTORIES

Treat	1	2	3	4
	1.360	1.679	0.985	1.655
Soil	Sand	Clay-Loam	Sandy Loam	Clay
	1.466	1.284	1.430	1.500
Hist	0	1	3	4
	1.376	1.352	1.554	1.398

SE (difference) treatments, soils and histories $\pm$ 0.0340 ***

Soil	Sand	Clay Loam	Sandy Loam	Clay	Mean
Harvest					
1	0.917	0.835	1.296	1.180	1.057
2	1.442	1.076	1.646	1.523	1.422
3	1.755	1.311	1.567	1.461	1.524
4	1.683	1.746	1.645	1.634	1.677
5	1.782	1.430	1.424	1.533	1.542
6	1.411	1.334	1.376	1.378	1.375
7	1.374	1.291	1.252	1.679	1.399
8	1.364	1.248	1.233	1.609	1.363
Treat					
1	1.495	1.069	1.480	1.396	1.360
2	1.700	1.530	1.652	1.833	1.679
3	0.940	1.048	0.845	1.106	0.985
4	1.729	1.488	1.741	1.663	1.655
Mean	1.466	1.284	1.430	1.500	1.420

SE (difference) soil v harvest $\pm$ 0.0961 ***
soil v treatment $\pm$ 0.0680 ***

EXPERIMENT 2: TAKE-ALL INFECTION (transformed data) HARVESTS 1 to 8, WITH EXPERIMENTAL TREATMENTS ON DIFFERENT SOIL TEXTURES AND CEREAL HISTORIES

	History				
	0	1	3	4	Mean
Harvest					
1	0.837	0.881	1.381	1.129	1.057
2	1.242	1.169	1.787	1.490	1.422
3	1.447	1.518	1.633	1.496	1.524
4	1.761	1.639	1.594	1.714	1.677
5	1.526	1.546	1.568	1.528	1.542
6	1.381	1.300	1.516	1.301	1.375
7	1.383	1.406	1.516	1.291	1.399
8	1.428	1.354	1.440	1.232	1.363
Treat					
1	1.330	1.258	1.508	1.346	1.360
2	1.596	1.611	1.872	1.637	1.679
3	0.971	0.933	1.044	0.991	0.985
4	1.606	1.605	1.793	1.617	1.655
Soil					
S	1.394	1.538	1.586	1.346	1.466
CL	1.234	1.287	1.317	1.296	1.284
SL	1.347	1.239	1.716	1.417	1.430
C	1.527	1.342	1.598	1.532	1.500
Mean	1.376	1.352	1.554	1.398	1.420

SE (difference) history v harvest $\pm$ 0.0961 ***
history v treatment $\pm$ 0.0680 ns
history v soil $\pm$ 0.0680 ***

EXPERIMENT 2: TAKE-ALL INFECTION (transformed data), HARVESTS 1 to 8, WITH EXPERIMENTAL TREATMENTS ON DIFFERENT SOIL TEXTURES AND CEREAL HISTORIES

	Treatment				
	1	2	3	4	Mean
Harvest					
1	1.016	1.138	0.936	1.137	1.057
2	1.530	1.696	0.870	1.592	1.422
3	1.561	1.941	0.816	1.777	1.524
4	1.767	2.094	0.778	2.068	1.677
5	1.549	1.846	0.874	1.900	1.542
6	1.303	1.649	0.917	1.629	1.375
7	1.122	1.627	1.210	1.637	1.399
8	1.034	1.443	1.476	1.501	1.363

SE (difference) treatment v harvest ± 0.0961 ***

		History			
Treat	Soil	0	1	3	4
1	S	1.365	1.571	1.540	1.505
	CL	1.054	1.129	1.154	0.941
	SL	1.347	1.253	1.850	1.470
	C	1.555	1.077	1.486	1.467
2	S	1.596	1.763	1.906	1.537
	CL	1.376	1.630	1.641	1.474
	SL	1.537	1.467	1.944	1.661
	C	1.876	1.583	1.999	1.876
3	S	0.909	1.077	0.962	0.812
	CL	1.108	0.844	1.009	1.230
	SL	0.734	0.709	1.105	0.834
	C	1.131	1.103	1.101	1.089
4	S	1.707	1.743	1.937	1.528
	CL	1.399	1.548	1.466	1.540
	SL	1.770	1.525	1.965	1.704
	C	1.547	1.604	1.804	1.695

SE (difference) treatment v soil v history ± 0.1360 ns

EXPERIMENT 2: TAKE-ALL INFECTION (transformed data), HARVESTS 1 to 8, WITH EXPERIMENTAL TREATMENTS ON DIFFERENT SOIL TEXTURES AND CEREAL HISTORIES

Harvest	Soil	History 0	1	3	4
1	S	0.780	0.939	1.220	0.729
	CL	0.863	0.812	0.800	0.863
	SL	0.830	0.912	2.031	1.411
	C	0.878	0.859	1.471	1.513
2	S	1.249	1.566	1.736	1.218
	CL	0.920	0.942	1.415	1.028
	SL	1.207	1.141	2.171	2.066
	C	1.590	1.025	1.828	1.649
3	S	1.645	1.932	1.815	1.628
	CL	1.178	1.372	1.345	1.350
	SL	1.581	1.488	1.758	1.440
	C	1.385	1.278	1.615	1.568
4	S	1.621	1.672	1.707	1.733
	CL	1.848	1.707	1.760	1.669
	SL	1.811	1.600	1.473	1.695
	C	1.763	1.577	1.436	1.758
5	S	1.693	1.912	1.856	1.669
	CL	1.242	1.728	1.228	1.522
	SL	1.575	1.211	1.600	1.309
	C	1.596	1.335	1.588	1.613
6	S	1.355	1.407	1.534	1.349
	CL	1.358	1.246	1.475	1.255
	SL	1.357	1.312	1.519	1.315
	C	1.455	1.236	1.534	1.286
7	S	1.355	1.341	1.497	1.301
	CL	1.189	1.358	1.293	1.324
	SL	1.262	1.187	1.497	1.063
	C	1.727	1.740	1.776	1.475
8	S	1.456	1.539	1.325	1.137
	CL	1.276	1.134	1.223	1.359
	SL	1.154	1.059	1.679	1.039
	C	1.824	1.683	1.533	1.394

SE (difference) history v soil v harvest $\pm$ 0.1922*

EXPERIMENT 2: TAKE-ALL INFECTION (transformed data), HARVESTS 1 to 8, WITH EXPERIMENTAL TREATMENTS ON DIFFERENT SOIL TEXTURES AND CEREAL HISTORIES

Harvest	Treat	Soil			
		Sand	Clay-Loam	Sandy-Loam	Clay
1	1	0.818	0.802	1.387	1.055
	2	1.010	0.765	1.397	1.380
	3	0.910	0.887	1.132	0.815
	4	0.930	0.883	1.267	1.470
2	1	1.542	1.169	1.846	1.564
	2	1.397	1.226	2.051	2.107
	3	0.959	0.912	0.700	0.910
	4	1.870	0.998	1.989	1.511
3	1	1.695	1.117	1.998	1.432
	2	2.153	1.772	1.800	2.038
	3	0.851	0.811	0.792	0.810
	4	2.321	1.545	1.677	1.565
4	1	1.917	1.818	1.705	1.628
	2	2.016	2.258	2.005	2.098
	3	0.796	0.773	0.783	0.761
	4	2.004	2.135	2.086	2.048
5	1	2.046	1.245	1.482	1.423
	2	2.156	1.612	1.575	2.042
	3	0.842	1.143	0.650	0.863
	4	2.086	1.721	1.988	1.804
6	1	1.371	1.011	1.469	1.363
	2	1.836	1.591	1.638	1.530
	3	0.940	1.163	0.660	0.906
	4	1.499	1.570	1.735	1.712
7	1	1.448	0.688	1.070	1.282
	2	1.598	1.639	1.361	1.909
	3	0.809	1.284	0.880	1.867
	4	1.640	1.552	1.698	1.657
8	1	1.126	0.704	0.883	1.421
	2	1.438	1.380	1.392	1.564
	3	1.413	1.408	1.167	1.916
	4	1.480	1.500	1.489	1.533

SE (difference) soil v treatment v harvest $\pm$ 0.1922 **

EXPERIMENT 2: TAKE-ALL INFECTION (transformed data), HARVESTS 1 to 8, WITH EXPERIMENTAL TREATMENTS ON DIFFERENT SOIL TEXTURES AND CEREAL HISTORIES

Harvest	Treat	History 0	1	3	4
1	1	0.823	0.848	1.189	1.202
	2	0.764	0.957	1.569	1.262
	3	0.905	0.814	1.103	0.922
	4	0.856	0.904	1.660	1.129
2	1	1.098	1.210	2.059	1.755
	2	1.568	1.311	2.189	1.715
	3	0.804	0.882	0.973	0.820
	4	1.497	1.271	1.929	1.671
3	1	1.500	1.527	1.699	1.517
	2	1.894	1.957	1.985	1.927
	3	0.741	0.734	1.044	0.745
	4	1.654	1.852	1.804	1.797
4	1	1.919	1.690	1.645	1.814
	2	2.088	2.047	2.030	2.212
	3	0.826	0.650	0.775	0.861
	4	2.211	2.169	1.925	1.969
5	1	1.751	1.358	1.585	1.501
	2	1.790	1.935	1.906	1.753
	3	0.766	0.919	0.740	1.072
	4	1.799	1.973	2.041	1.786
6	1	1.364	1.226	2.498	2.126
	2	1.539	1.541	1.894	1.620
	3	0.957	0.817	1.013	0.882
	4	1.664	1.618	1.657	1.577
7	1	1.185	1.006	1.247	1.052
	2	1.605	1.724	1.839	1.339
	3	1.269	1.111	1.247	1.213
	4	1.474	1.784	1.730	1.559
8	1	1.000	1.197	1.138	0.798
	2	1.524	1.414	1.566	1.270
	3	1.496	1.537	1.457	1.415
	4	1.690	1.268	1.599	1.445

SE (difference) history v treatment v harvest $\pm$ 0.1922 ns

EXPERIMENT 3: THE EFFECT OF SOIL SERIES, HISTORY AND CULTIVATION ON TAKE-ALL DEVELOPMENT OVER 8 HARVESTS
(cm infected root per root)

	HARVEST								
	1	2	3	4	5	6	7	8	Mean
Soil									
HK	0.3	3.0	5.0	4.4	3.6	3.9	3.5	3.2	3.4
ME	0.3	3.2	5.3	4.7	4.4	4.7	5.0	4.5	4.0
WN	0.2	3.3	4.3	4.7	4.8	5.6	5.3	5.2	4.2
SE (difference)	0.90	0.60	0.64	0.62	0.58	0.57	0.56	0.53	
	ns	ns	ns	ns	ns	*	*	***	
history									
0	0.1	1.3	4.0	4.8	4.7	4.5	4.6	4.0	3.5
4	0.4	3.7	5.5	4.4	3.6	4.3	4.2	3.6	3.7
8	0.2	3.6	4.9	4.5	3.8	4.9	4.4	4.3	3.8
11/12	0.3	4.1	5.1	4.7	5.1	5.2	5.4	5.4	4.4
SE (difference)	0.10	0.70	0.73	0.70	0.68	0.65	0.64	0.49	
	ns	**	ns	ns	ns	ns	ns	*	
Cultivation									
-	-	4.1	5.9	5.2	4.8	5.6	5.4	4.7	5.1
+	-	2.3	3.9	4.1	3.9	4.0	3.9	4.0	3.7
SE (difference)	-	0.49	0.52	0.50	0.48	0.47	0.45	0.35	
		**	***	*	ns	**	**	ns	
Mean	0.3	3.2	4.9	4.6	4.3	4.7	4.7	4.3	3.9

EXPERIMENT 3: THE EFFECT OF SOIL SERIES, HISTORY AND CULTIVATION ON TAKE-ALL DEVELOPMENT OVER 8 HARVESTS
(cm infected root per root)

		HARVEST							
		2	3	4	5	6	7	8	Mean
Soil	Cultivation								
HK	-	3.5	6.4	4.9	4.0	5.2	4.4	3.2	4.5
	+	2.5	3.8	4.0	3.3	2.8	2.5	3.3	3.2
ME	-	4.1	6.5	5.3	4.8	6.4	6.3	4.9	5.5
	+	2.4	4.2	4.2	4.1	3.4	3.8	4.2	3.8
WN	-	4.7	4.8	5.5	5.5	5.2	5.3	6.0	5.3
	+	1.9	3.8	4.0	4.2	6.1	5.2	4.4	4.2
SE (difference)		0.84	0.90	0.88	0.85	0.80	0.79	0.60	
		ns	ns	ns	ns	**	ns	ns	
History									
0	-	1.8	5.4	5.0	4.5	4.7	4.6	5.1	4.4
	+	0.7	2.9	4.6	4.9	4.4	4.5	3.0	3.6
4	-	3.6	6.7	4.8	5.8	5.5	5.3	4.6	5.2
	+	3.7	4.5	4.1	1.7	3.2	3.2	2.5	3.3
8	-	5.2	5.6	5.3	3.4	6.3	5.1	3.6	4.9
	+	2.2	4.2	3.8	4.1	3.8	3.8	5.1	3.9
11/12	-	5.8	6.1	5.7	5.4	5.9	6.6	5.5	5.9
	+	2.5	4.2	3.8	4.7	4.7	4.2	5.2	4.2
SE (difference)		0.98	1.05	1.02	0.98	0.95	0.92	0.70	
		ns	ns	ns	*	ns	ns	**	
Mean		3.2	4.9	4.6	4.3	4.7	4.7	4.3	4.4

EXPERIMENT 3: THE EFFECT OF SOIL SERIES, HISTORY AND CULTIVATION ON TAKE-ALL DEVELOPMENT OVER 8 HARVESTS
(cm infected root per root)

Harvest	Soil	Cultivation	history (years in cereals) 0	4		8		11/12		SE (difference) S v H	S v H v C
1	HK		0.3		0.5		0.4		0.2		
	ME		0.1		0.7		0.2		0.4	0.18 ns	
	WN		0.1		0.1		0.1		0.5		
2	HK	- 1.4	0.9	3.9	3.4	3.9	3.4	4.6	4.2		
		+ 0.4		2.8		2.9		3.5			
	ME	- 3.2	2.2	1.9	3.6	4.3	2.6	6.7	4.5	1.23 ns	1.92 ns
		+ 1.3		5.0		1.0		1.9			
	WN	- 0.3	0.3	4.8	4.0	7.4	4.9	6.0	3.7		
		+ 0.3		3.2		2.6		1.4			
3	HK	- 7.3	3.8	6.8	5.2	5.1	4.8	6.3	6.2		
		+ 0.7		3.7		4.6		5.9			
	ME	- 5.4	5.8	8.0	6.0	5.9	4.9	6.4	4.3	1.27 ns	1.76 ns
		+ 5.6		4.2		4.0		2.5			
	WN	- 2.7	2.0	5.2	5.4	6.1	5.0	5.4	4.7		
		+ 1.4		5.6		4.0		4.1			
4	HK	-4.7	4.1	3.8	4.5	3.4	3.8	7.5	5.3		
		+ 3.7		5.0		4.2		3.2			
	ME	- 4.9	5.1	6.2	4.6	5.6	5.1	4.4	3.9	1.25 ns	1.86 ns
		+ 5.3		3.3		4.7		3.6			
	WN	- 5.5	5.3	4.0	4.1	6.7	4.6	4.9	4.8		
		+ 5.1		4.0		2.3		4.4			
5	HK	- 3.6	3.5	5.8	3.8	2.0	2.8	4.6	4.5		
		+ 3.4		2.1		3.5		4.4			
	ME	- 5.3	4.8	5.7	3.9	1.4	2.4	7.4	6.8	1.19 *	1.87 ns
		+ 4.5		2.5		3.4		6.1			
	WN	- 4.7	5.8	6.4	3.0	7.5	6.5	4.0	3.8		
		+ 6.8		0.5		5.7		3.5			

EXPERIMENT 3: THE EFFECT OF SOIL SERIES, HISTORY AND CULTIVATION ON TAKE-ALL DEVELOPMENT OVER 8 HARVESTS
(cm infected root per root)

Harvest	Soil	Cultivation	history (years in cereals) 0		4		8		11/12		SE (difference) S v H	S v H v C
6	HK	- 5.1	3.9	4.2	4.0	5.6	3.3	6.2	4.4			
		+ 3.1		3.8		1.5		2.9				
	ME	- 5.7	3.8	6.8	3.9	7.9	5.6	5.3	5.7		1.13 ns	1.72 *
		+ 2.3		1.4		3.8		6.0				
	WN	- 3.6	6.0	5.4	4.9	5.5	6.0	6.4	5.7			
		+ 8.4		4.3		6.5		5.3				
7	HK	- 3.5	3.0	4.4	3.5	3.4	2.8	6.3	4.7			
		+ 2.5		2.6		1.9		3.0				
	ME	- 5.9	5.1	6.4	5.0	5.6	4.1	7.2	5.9		1.16 ns	1.74 ns
		+ 4.4		3.6		2.7		4.8				
	WN	- 4.2	5.4	4.9	4.1	5.8	6.4	6.4	5.5			
		+ 6.5		3.2		6.6		4.6				
8	HK	- 2.4	3.5	4.0	2.6	2.1	2.0	4.2	4.8			
		+ 4.5		1.2		1.8		5.4				
	ME	- 7.2	5.5	3.8	3.1	2.7	3.8	5.9	5.8		0.85 ***	1.21 *
		+ 3.9		2.5		4.8		5.7				
	WN	- 5.6	3.1	6.1	5.0	5.9	7.3	6.4	5.5			
		+ 0.5		3.9		8.7		4.5				

EXPERIMENT 4a: THE DEVELOPMENT OF TAKE-ALL INFECTION OVER 8 HARVESTS IN SOIL SAMPLES OF MISCELLANEOUS TEXTURES AND CEREAL HISTORIES (cm infected root per root)

Soil series	History	harvest 1	2	3	4	5	7	8	Mean
WN	0	0.0	3.7	3.6	2.7	1.8	2.5	3.1	2.5
	1	0.0	4.0	2.3	4.0	3.7	2.4	4.1	2.9
	2	3.0	2.6	8.4	3.5	3.2	3.9	3.7	4.0
	3	< 0.1	2.4	4.6	3.1	1.2	3.0	2.8	2.4
	4	0.1	0.1	4.8	1.7	2.7	3.6	3.8	2.4
	5	0.7	3.3	5.0	2.7	1.5	1.6	1.0	2.3
Mean		0.6	2.7	4.8	2.9	2.3	2.8	3.1	2.8
B1	0	0.0	2.5	1.8	5.5	3.9	4.9	3.5	3.2
	1	0.3	1.4	2.1	3.6	2.5	3.6	2.4	2.3
	2	1.4	6.9	3.7	4.8	3.6	5.6	2.2	4.0
	3	1.1	7.1	3.6	2.8	2.6	4.5	2.7	3.5
Mean		0.7	4.5	2.8	4.2	3.1	4.6	2.7	3.2

EXPERIMENT 4a: THE DEVELOPMENT OF TAKE-ALL INFECTION OVER 8 HARVESTS IN SOIL SAMPLES OF MISCELLANEOUS TEXTURES AND CEREAL HISTORIES (cm infected root per root)

Soil series	History	harvest 1	2	3	4	5	7	8	Mean
AL	0	0.0	1.1	5.7	5.7	6.5	3.5	3.8	3.7
	1	< 0.1	0.4	5.1	5.2	4.7	3.8	2.5	3.1
	2	1.1	3.5	4.7	4.4	3.9	3.3	2.2	3.3
DR	0	0.1	1.2	0.8	4.1	2.6	0.9	0.9	1.5
	1	0.3	1.4	8.4	4.0	4.0	1.5	0.6	2.9
	2	2.8	2.3	4.0	1.8	1.6	1.3	0.1	2.0
YA	0	0.0	0.2	1.8	3.4	1.7	4.1	3.8	2.1
	1	0.0	0.2	5.1	2.7	2.1	4.0	4.0	2.6
	2	0.6	0.5	2.6	2.3	2.8	4.2	4.8	2.7
MA	0	0.0	0.0	5.0	4.1	4.3	1.3	2.8	2.5
	1	0.3	2.0	5.9	3.2	5.0	3.6	3.1	3.3
	2	0.6	3.0	6.0	3.1	2.9	2.1	2.7	2.9
DL	0	0.0	1.0	1.8	2.9	3.8	3.6	2.8	2.3
	2	0.2	2.0	2.0	3.1	4.4	3.5	1.4	2.4
	4	0.7	4.1	3.1	4.9	6.3	3.4	2.9	3.6
CS	0	0.0	0.2	4.1	4.6	5.1	3.7	6.4	3.4
	1	1.1	2.1	8.0	3.4	3.5	2.6	7.2	4.0
BE	0	0.9	0.1	4.4	3.0	4.4	2.3	5.8	3.0
	2	7.5	3.6	7.8	4.2	3.1	3.9	5.8	5.1
ME	1	0.2	< 0.1	1.7	1.7	0.5	1.3	0.1	0.8
	3	0.0	0.4	4.2	1.8	1.8	3.0	3.3	2.1
HK	0	0.0	3.5	1.7	4.5	1.8	3.9	3.3	2.7
FR	2	0.6	1.4	5.4	3.9	1.5	0.7	0.4	2.0
PF	3	0.3	0.8	5.3	3.3	1.3	0.1	1.3	1.7
KK	5	0.1	1.0	0.7	0.7	2.2	0.7	0.2	0.8

EXPERIMENT 4b: THE DEVELOPMENT OF TAKE-ALL INFECTION OVER 8 HARVESTS IN SOIL SAMPLES OF MISCELLANEOUS TEXTURES AND NON-CEREAL HISTORY (cm infected root per root)

Soil series	harvest 1	2	3	4	5	6	7	8	Mean
AL	< 0.1	0.8	5.9	5.9	5.9	5.8	6.9	3.4	4.3
AL	< 0.1	1.6	5.1	4.5	4.6	5.1	4.0	2.7	3.5
DR	< 0.1	0.9	1.4	4.1	6.1	5.0	4.1	4.0	3.2
DR	< 0.1	0.2	3.7	1.3	6.2	6.1	5.2	3.4	3.3
EK	0.0	0.5	1.9	6.0	5.3	5.8	4.8	3.9	3.5
PF	0.0	0.2	0.1	0.9	2.7	3.1	3.2	6.6	2.1
HK	0.1	0.6	5.8	6.7	2.2	3.9	5.1	2.9	3.4
DL	< 0.1	0.2	< 0.1	< 0.1	2.7	0.4	0.0	0.3	0.5
YA	0.2	4.6	5.8	8.2	7.0	6.0	4.0	4.0	5.0
WN	< 0.1	0.4	1.2	2.2	3.5	1.6	5.1	1.1	1.9
KK	0.0	0.4	2.3	0.3	7.0	6.2	6.1	3.4	3.2
BE	< 0.1	< 0.1	0.8	4.8	2.5	6.5	3.9	3.1	2.7
CU	< 0.1	0.9	0.9	2.5	3.1	4.8	5.9	5.3	2.9
HM	< 0.1	0.4	2.7	5.9	1.9	4.1	4.6	1.4	2.6
WH	0.0	0.1	0.1	0.1	0.1	2.8	1.7	1.4	0.7

DETAILS OF THE SOIL SERIES REFERRED TO IN THE TEXT

Soil association	Soil series			
	Drainage			
	free	imperfect	poor	Topsoil texture
Biel		Biel		clay-loam
Darleith	Darleith			loam
Dreghorn	Dreghorn			sand
		Peffer		sand
Eckford	Eckford			sand
Fraserburgh	Fraserburgh			sand
Hobkirk	Hobkirk			sandy loam
			Cessford	clay loam
Humbie		Humbie		sandy clay loam
		Morham		sandy loam
Kilmarnock		Kilmarnock		clay loam
Minto		Belses		clay loam
Stirling		Cauldside		silty clay
Whitsome		Whitsome		clay
			Horndean	clay loam
Winton		Winton		sandy clay loam
		Macmerry		sandy loam
Yarrow	Yarrow			sandy loam
	Alluvium			sand

APPENDIX III

EXPERIMENT 1a: ANALYSIS OF DATA

OBSERVED

Soil series	Years in cereals	BOUNDS 1.0	2.0	3.0	6.0 >	6.0 cm	Total
HK	0	3	5	4	13	7	32
WN	0	1	7	8	12	4	32
KK	0	5	4	5	5	2	21
PF	0	2	4	3	3	3	15
CU	0	6	8	7	9	1	31
HM	0	3	13	7	7	6	36
EK	0	9	11	3	6	0	29
WH	0	6	10	9	18	4	47
YA	0	4	2	7	14	7	34
BE	0	6	5	4	24	18	57
AL	0	8	4	4	7	3	26
AL	0	3	3	5	26	8	45
DR	0	3	12	6	17	2	40
DR	0	4	6	7	11	3	31
	Mean	63	94	79	172	68	476

EXPECTED

Soil series	Years in cereals	BOUNDS 1.0	2.0	3.0	6.0 >	6.0 cm	chi
HK	0	4.2	6.3	5.3	11.6	4.6	2.43
WN	0	4.2	6.3	5.3	11.6	4.6	3.99
KK	0	2.8	4.1	3.5	7.6	3.0	3.65
PF	0	2.0	3.0	2.5	5.4	2.1	1.89
CU	0	4.1	6.1	5.1	11.2	4.4	5.21
HM	0	4.8	7.1	6.0	13.0	5.1	8.63
EK	0	3.8	5.7	4.8	10.5	4.1	18.54***
WH	0	6.2	9.3	7.8	17.0	6.7	1.41
YA	0	4.5	6.7	5.6	12.3	4.9	4.88
BE	0	7.5	11.3	9.5	20.6	8.1	19.44***
AL	0	3.4	5.1	4.3	9.4	3.7	7.86
AL	0	6.0	8.9	7.5	16.3	6.4	12.40*
DR	0	5.3	7.9	6.6	14.5	5.7	6.05
DR	0	4.1	6.1	5.1	11.2	4.4	1.14

From a 5 x 14 table chi = 96.71174***

DF = 52

EXPERIMENT 1b: ANALYSIS OF DATA

OBSERVED

Soil series	Years in cereals	BOUNDS 1.0	2.0	3.0	6.0	> 6.0 cm	Total
KK	1	6	9	8	6	2	31
KK	5	5	10	1	5	1	22
EK	2	10	9	2	4	0	25
EK	2	10	8	1	8	0	27
EK	4	4	7	5	11	1	28
EK	5	7	12	10	12	5	46
AL	2	2	8	20	23	17	70
HK	2	10	5	3	3	0	21
BE	2	3	6	3	8	0	20
CU	2	2	10	5	29	3	49
DL	3	4	6	10	13	5	36
WN	3	3	7	2	16	1	29
WN	8	11	6	6	18	0	41
WH	5	3	4	4	5	0	16
HE	5	9	12	9	32	5	67
CS	5	1	5	8	27	3	44
Mean		90	124	97	220	41	577

EXPECTED

Soil series	Years in cereals	BOUNDS 1.0	2.0	3.0	6.0	> 6.0 cm	chi
KK	1	4.9	6.7	5.3	11.9	2.2	5.43
KK	5	3.5	4.8	3.7	8.5	1.6	10.05*
EK	2	3.9	5.4	4.2	9.6	1.8	17.98**
EK	2	4.2	5.9	4.6	10.4	1.9	13.85**
EK	4	4.4	6.1	4.7	10.8	2.0	0.70
EK	5	7.2	10.0	7.8	17.7	3.3	3.75
AL	2	11.0	15.2	11.9	26.9	5.0	45.52***
HK	2	3.3	4.6	3.6	8.1	1.5	18.40**
BE	2	3.1	4.3	3.4	7.7	1.4	2.14
CU	2	7.7	10.6	8.3	18.8	3.5	11.13*
DL	3	5.7	7.8	6.1	13.8	2.6	3.51
WN	3	4.6	6.3	4.9	11.2	2.1	5.01
WN	8	6.5	8.9	7.0	15.8	2.9	7.53
WH	5	2.5	3.5	2.7	6.2	1.1	2.15
HE	5	10.5	14.5	11.4	25.8	4.8	2.67
CS	5	6.9	9.5	7.5	16.9	3.2	13.27*

From a 5 x 16 table chi = 163.09239***

DF = 60

EXPERIMENT 2: ANALYSIS OF DATA

OBSERVED

Treatment	Soil series	Years in cereals	BOUNDS 1.0	2.0	3.0 >	3.0 cm	Total
Cult	HK	0	14	5	7	7	33
Cult	HK	4	9	8	7	8	32
Cult	HK	8	3	1	2	3	9
Cult	HK	12	6	5	10	18	39
Non Cult	HK	0	8	9	1	7	25
Non Cult	HK	4	4	3	1	2	10
Non Cult	HK	8	6	5	4	4	19
Non Cult	HK	12	5	7	1	2	15
Cult	WN	0	2	1	0	13	16
Cult	WN	4	3	2	5	21	31
Cult	WN	8	1	4	5	17	27
Cult	WN	11	14	7	1	6	28
Non Cult	WN	0	5	7	3	11	26
Non Cult	WN	4	5	2	2	10	19
Non Cult	WN	8	4	3	7	29	43
Non Cult	WN	11	2	3	4	9	18
Cult	ME	0	3	11	8	20	42
Cult	ME	4	9	10	4	14	37
Cult	ME	8	2	4	2	6	14
Cult	ME	11	3	11	6	7	27
Non Cult	ME	0	4	4	8	21	37
Non Cult	ME	4	3	6	2	16	27
Non Cult	ME	8	8	11	4	15	38
Non Cult	ME	11	4	10	3	13	30
	Mean		113	124	91	266	594

OBSERVED

Soil series	BOUNDS 1.0	2.0	3.0 >	3.0 cm	Total
HK	32	19	26	36	113
WN	23	24	7	15	69
ME	20	14	11	57	102
Mean	75	57	44	108	284

EXPERIMENT 2: ANALYSIS OF DATA (continued)

EXPECTED

Treatment	Soil series	Years in cereals	BOUNDS 1.0	2.0	3.0	> 3.0	chi
Cult	HK	0	6.3	6.9	5.1	14.8	14.86**
Cult	HK	4	6.1	6.7	4.9	14.3	5.35
Cult	HK	12	7.4	8.1	6.0	17.5	4.21
Non Cult	HK	0	4.8	5.2	3.8	11.2	8.62*
Non Cult	HK	8	3.6	4.0	2.9	8.5	4.64
Cult	WN	0	3.0	3.3	2.5	7.2	9.20*
Cult	WN	4	5.9	6.5	4.7	13.9	8.18*
Cult	WN	8	5.1	5.6	4.1	12.1	5.98
Cult	WN	11	5.3	5.8	4.3	12.5	20.28***
Non Cult	WN	0	4.9	5.4	4.0	11.6	0.73
Non Cult	WN	4	3.6	4.0	2.9	8.5	2.05
Non Cult	WN	8	8.2	9.0	6.6	19.3	11.07*
Non Cult	WN	11	3.4	3.8	2.8	8.1	1.41
Cult	ME	0	8.0	8.8	6.4	18.8	4.14
Cult	ME	4	7.0	7.7	5.7	16.6	2.11
Cult	ME	11	5.1	5.6	4.1	12.1	8.98*
Non Cult	ME	0	7.0	7.7	5.7	16.6	5.25
Non Cult	ME	4	5.1	5.6	4.1	12.1	3.28
Non Cult	ME	8	7.2	7.9	5.8	17.0	2.08
Non Cult	ME	11	5.7	6.3	4.6	13.4	3.31

EXPECTED

Soil series	BOUNDS 1.0	2.0	3.0	> 3.0	chi
HK	29.8	22.7	17.5	43.0	6.00
WN	18.2	13.8	10.7	26.2	14.78**
ME	26.9	20.5	15.8	38.8	13.84**

From a 4 x 3 Table chi = 34.62872***

DF = 6

EXPERIMENT 3: ANALYSIS OF DATA

OBSERVED

Treatment	N level	BOUNDS 1.5	3.0	4.0	> 4.0 cm	Total
A	Low	22	19	12	14	67
A	High	26	16	4	14	60
B	Low	37	32	20	38	127
B	High	45	40	19	7	111
C	Low	31	30	10	25	96
C	High	14	20	8	9	51
D	Low	14	18	8	12	52
D	High	7	9	3	6	25
	Mean	196	184	84	125	589

EXPECTED

Treatment	N level	BOUNDS 1.5	3.0	4.0	> 4.0 cm	chi
A	Low	22.3	20.9	9.6	14.2	0.81
A	High	20.0	18.7	8.6	12.7	4.78
B	Low	42.3	39.7	18.1	27.0	6.86
B	High	36.9	34.7	15.8	23.6	14.85***
C	Low	31.9	30.0	13.7	28.4	2.07
C	High	17.0	15.9	7.3	10.8	1.94
D	Low	17.3	16.2	7.4	11.0	0.95
D	High	8.3	7.8	3.6	5.3	0.57

From a 4 x 8 table chi = 32.83643*

DF = 21

EXPERIMENT 4: ANALYSIS OF DATA

OBSERVED

Soil series	Years in cereals	Assay crop	BOUNDS 2.5	4.5	> 4.5 cm	Total
ME	0	W	11	10	3	24
		B	4	18	15	37
ME	8	W	14	15	8	37
		B	6	5	0	11
ME	11	W	7	7	5	19
		B	9	7	0	16
WN	11	W	5	6	3	14
		B	4	18	2	24
YA	0	W	7	21	10	38
		B	8	9	16	33
AL	0	W	5	7	11	23
		B	3	12	15	30
WH	0	W	5	13	10	28
		B	1	10	10	21
		Mean	78	147	105	330

EXPECTED

Soil series	Years in cereals	Assay crop	BOUNDS 2.5	4.5	> 4.5 cm	chi
ME	0	W	5.7	10.7	7.6	7.86*
		B	8.7	16.5	11.8	3.60
ME	8	W	8.7	16.5	11.8	4.50
ME	11	W	4.5	8.5	6.0	1.84
		B	3.8	7.1	5.1	12.29**
WN	11	B	5.7	10.7	7.6	9.65**
YA	0	W	9.0	16.9	12.1	1.78
		B	7.8	14.7	10.5	5.10
AL	0	W	5.4	10.2	7.3	2.92
		B	7.1	13.4	9.5	5.62
WH	0	W	6.6	12.5	8.9	0.55
		B	5.0	9.4	6.7	4.86

From a 3 x 12 table chi = 60.55633***

DF = 22

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